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VOLUME 10 DECEMBER, 1929 NUMBER 6

PLASTIC SURGERY OF THE EAR, NOSE AND FACE*

ALBERT D. DAVIS, D.D.S., M.D.

SAN FRANCISCO

Though plastic surgery showed great development as the result of necessity during the World War, its origin dates back to the time of earliest antiquity. Researches by Mauclaire, of the medical faculty of the University of Paris, reveal histories of the priest-surgeons of India and cases of rhinoplasty as early as 800 B.C. These cases were the result of the custom at that time of cutting off the nose as a punishment for theft. The operation consisted of peeling down a skin flap from the forehead, leaving the lower end attached and shaping it into a rough semblance of a nose. Such punitive mutilations were common during those ancient times.

In 1440 A.D. the Brancas, noted surgeons of their day, developed the Italian method of rhinoplasty. Narrow strips of skin cut from the inner surface of the arm were used to restore lost noses, the arm being immured in a fixation apparatus for a period of fourteen days, when the connection of the graft was severed and shaped into a nose. A little later, a Viennese surgeon, Tagliacozzi, invented an elaborate but effective device for holding the arm in contact with the nose during the healing process. A picture of his apparatus was printed from a woodcut in his book on "Surgery" in 1597.

As time passed, surgeons became more daring, and in 1751 one finds recorded the report of a surgeon who had replaced a nose that had been cut off in a duel and had fallen in the mud. A humorous incident followed. When he reported the success of his operation he was almost ostracized by his confrères for presuming to report such an unbelievable occurrence.

During these stirring times dueling was at its height, and numerous cases came under observation. It is not difficult to understand the psychic effect on the gentleman who had lost his nose on the "field of honor" when a thief was similarly mutilated as a punishment for his crime. Nor is it incomprehensible to assume that he would endeavor in every way possible to have the lost organ restored. If now and then

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* From the Department of Plastic Surgery, Leland Stanford University School of Medicine.

a prosperous thief engaged the services of the surgeon and profited thereby, interest in this particular type of surgery would be stimulated rather than diminished.

To refer again to the early plastic operations, Celsus (37 B.C. to 42 A.D.) reported his method of an operation for cheiloplasty. Dr. John Hunter of England carried on some rather extensive experiments in animals during his active years, transplanting not only tissues but entire organs from one animal to another. Still later, the brilliant work of Ollier, Hamilton, Warren, Netolilski, Reverdin, Thiersch, Wolfe, Krause and others stimulated interest in plastic surgery, and rapid strides were made.

During the World War, necessity often prompted the invention of new ideas, and with the recognition of the specialists by the medical corps of the army, groups of men particularly interested in and adapted to plastic surgery developed an almost standardized technic.

OTOPLASTY

Probably the most difficult of all reconstructive operations is the restoration of the ear, whether the lesion is congenital or acquired. Even minor lesions require the utmost care, and the end-results are often disappointing. The prominence of the location and the conspicuousness of the deformity, especially in the male sex, present a problem that calls forth patience, skill and artistry.

Even so, while the work may progress slowly and the result may not be as perfect as one might wish, a reconstructed ear from the patient's own tissues is a step far in advance of a prosthesis of celluloid, rubber, papier maché or other material held in place by adhesive tape, notwithstanding statements of authors to the contrary.

Szymanowski was one of the first to outline a definite technic in the restoration of an auricle. His procedure consisted of raising flaps from the scalp and mastoid region, folding the tissue on itself and suturing the margins, later grafting the uncovered portion of the defect.

The use of cartilage transplants from the rib insures a fair degree of success in the operation, but the difficulty lies in maintaining the auricle flap in its proper position. Modifications of Szymanowski's technic are still in use.

Gillies of London took cartilage from the rib, shaped and cut it to form, raised a flap from the scalp and inserted the cartilage. Then with modeling wax or Stent's composition, the flap was pushed into the folds and grooves of the cartilage and held in place until healing occurred. When flaps were not available, owing to trauma, a tubed pedicle from the neck or arm was first transplanted to the auricular site.

The best solution to auricular reconstructions are modifications of these methods offered by Pierce of San Francisco and by Graham, who uses a slightly different technic. Pierce's method consists of the primary implantation of cartilage with a base long enough to retain it in position when raised. Six weeks later, a new incision outlining the antihelix is made, which is then raised and a Thiersch graft on Stent composition inserted so as to cover the entire raw area. The edges are sutured back into place, leaving the Stent composition in the center; this is removed in ten days. In the meantime, a tubed pedicle is made and stepped up to place to form the new helix. Later trimming and adjusting allow a good result.

Fig. 1.—Complete destruction of nose of syphilitic origin. A lined forehead flap of proper dimensions will be necessary for reconstruction followed by a cartilage implant for support.

Graham used septal cartilage from the nose transplanted above and behind the defect; four weeks later, a circular incision is made beginning in the upper anterior part of the ear; it is carried to the periosteum and includes in it the cartilage transplant. The tissues thus raised are folded in such a way as to have the upper border of the ear covered by a continuous layer of skin. The anterior portion is then sutured down to the tragus and the scalp defect grafted. A month later, the lower part is incised, carried forward and sutured to the freshened lobule; another graft is placed posteriorly and a few minor trimmings made to obtain better contour.

Of the methods given, I prefer the technic outlined by Pierce, for the reason that the reconstructed auricle may be made of the same size and more nearly of the same design as the normal one.

Fig. 2.—Congenital saddle nose deformity, before and after operation. A Thiersch graft was employed, supported by a cartilage implant from the rib.

Fig. 3.—Typical "bird-beak" deformity due to fracture, before and after operation.

Tissue taken from other parts of the body for reconstruction of the auricle is of different consistency and appearance from that of the normal ear, and is therefore used only when scalp tissue is unavailable. Celluloid and other foreign bodies are to be condemned in favor of cartilage as a permanent support.

Minor defects of the ear are usually corrected with little difficulty. A number of operations designed by various surgeons cover adequately the more common types of deformities.

RHINOPLASTY

One of the most interesting as well as the most common deformities with which the plastic surgeon deals is the correction of deformities of the nose. Occupying as it does such a prominent place on the face, the nose offers a difficult problem in cosmetic repair even though the defect may seem to be quite simple. For the same reason, the nose is subject to trauma more than any other part of the face.

In a paper of this length it is impossible to discuss all of the defects that occur. Congenital malformations, disease and trauma all play a part in deformities of the nose, but one rule applies in their correction which is universal in application to all mucus-lined cavities, namely, "If the mucosa is involved in the deformity, it must be replaced by inturned flaps or by skin grafts if there is a defect." Attempts at restoration with a total disregard for the lining result in disappointment. Tunneled flaps are of great value in minor defects in the upper parts of the nose, while larger losses require the use of the forehead or temporal artery flap.

The nasal bridge lends itself well to the application of either a Thiersch or a Wolfe graft, but the Wolfe graft may be taken from some portion of the face and is advantageous in that the texture and color of the skin are more harmonious than in that of a Thiersch graft from the arm or leg. Incisions for nasal plastic operations should be made as inconspicuous as possible. The "columellar" lift of Gillies is rarely necessary; the midcolumellar incision answers most of the needs for midnasal approach. Incisions in the glabellar region are used when forehead flaps have been previously turned down for major defects.

Total cosmetic reconstructive rhinoplasty is rare and infinite patience and cooperation with the patient are required if a good result is to be obtained.

When the framework of the nose has been destroyed, it must be reestablished and a lining membrane provided. Saddle nose, whether specific in origin or acquired, varies from a slight defect to a great loss, and a correct diagnosis is imperative.

Practically all cases of syphilitic saddle nose require the replacement of the lining. Cases in which the deformity is confined to the bridge of the nose and is due to the loss of the cartilaginous support are uncommon and require only the implantation of cartilage of the shape and size originally given to the nose by the septum. The common type of syphilitic nose requires both lining and support. In order to retain the skin graft in position, a removable cap splint is made on the teeth prior to operation. To the center of this splint a removable wire upright, long enough to cover the raw area, is attached.

At the first operation, an incision is made in the labial sulcus from one canine fossa to the other. The tissues are dissected up, and the nose is freed to the glabellar region mesially and laterally to the region of the inner canthus on each side.

Fig. 4.—Cut caused by broken windshield, one of the problems in accident cases. *A* shows the wounds which were closed at an emergency station immediately after the injury. There was a big loss of mucous lining of the cheek. An intra-oral skin graft was made with an appliance on the teeth holding a removable tray containing the Stent mold of the defect. Readjustment of vermilion border and scar excision followed. *B* shows results after three months.

The amount of undercutting necessary may best be judged by pulling gently on the tip of the nose until the shape wanted may be produced. Dental modeling compound is used to obtain an impression of the raw area, and after the compound is molded to form, a graft is cut and stretched firmly over the whole surface of the mold corresponding to the raw area. The upright is replaced, and the graft and mold together are inserted without disturbing the position of the graft. At the end of ten days the mold is removed; the cavity is washed with physiologic solution of sodium chloride, and either a fresh mold or the same one cleaned is reinserted into the cavity, which is now lined with skin.

The patient wears this appliance in an overcorrected position for eight weeks (to allow full contraction and solidity of the graft), after which a hinged cartilage is inserted between the graft and the outer skin of the nose, the appliance being discarded. Later, the oronasal sinus is closed. This is the operation as outlined by Gillies in 1922 and pub-

Fig. 5.—Cosmetic meloplasty before and after operation.

lished in 1923. The advantages of this method over others are that the scars are concealed, the results are more perfect, and all semblance of saddle nose is eradicated.

When destruction of the outer skin of the nose is present, in addition to loss of support and lining, a more or less extensive rhinoplasty is necessary, a forehead flap being used if feasible on account of the more harmonious color and texture of the skin.

Among the more common types of plastic operations on the nose is the removal of a hump. A more or less accurate estimate of the amount of tissue to be removed is necessary. In many cases the nasal bones must be fractured and displaced mesially to overcome the width of the bridge following removal of cartilage.

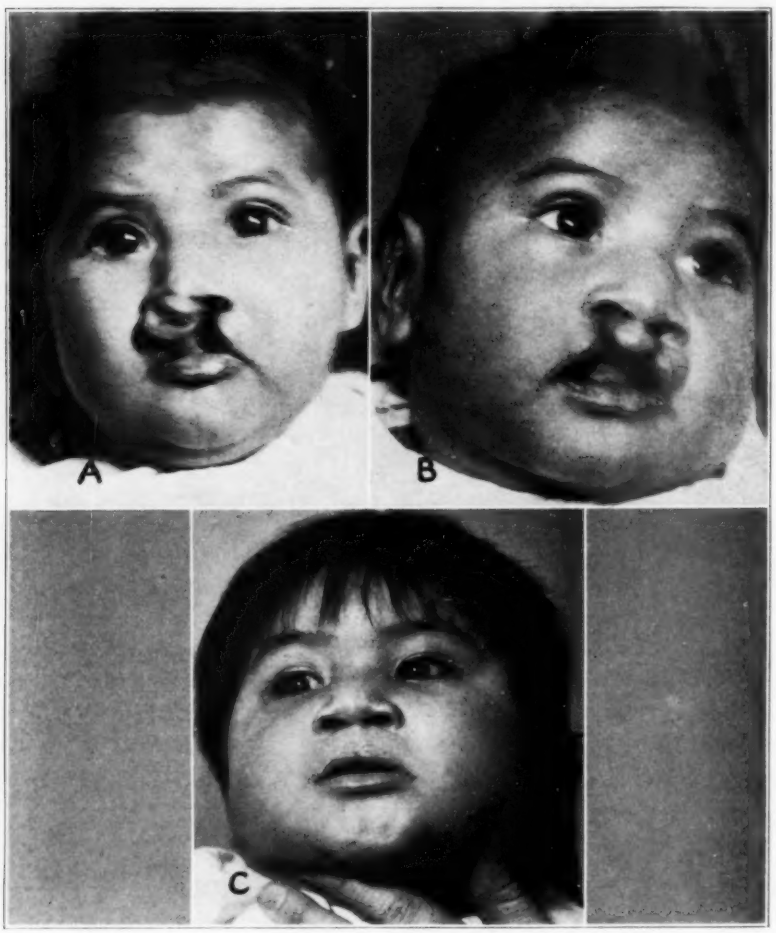


Fig. 6.—Congenital tripartite cleft lip and palate. *A* shows the position of the prolabiam and premaxillary bones. In *B*, the premaxillary bones have been placed in proper position, the edges freshened and brought into contact with the maxillary bones on each side of the cleft. Immobilization was secured by means of wire sutures. The prolabium and nose are in midline. This is the foundation on which a normal lip and nose may be constructed. These clefts are treated as ununited fractures. Mucoperiosteal flaps are raised at the point of contact and sutured over the bones. A normal dental arch is assured. In *C*, the cleft in the lip has been closed. The nose tip must be raised later.

FACIAL PLASTIC OPERATIONS

Patients who have facial deformities, whether comparatively trivial or grossly conspicuous, are, more often than not, so conscious of their disfigurement that they become depressed mentally and develop an inferiority complex which hinders them throughout life. Social and economic possibilities of these people are limited, and often grave psychoses with suicidal tendencies develop.

As no two cases are precisely similar, an accurate diagnosis is of paramount importance, and the procedure to be adopted should be carefully decided on before the work is attempted.

The operative work is exacting in the extreme; the after-care is of the utmost importance and often is prolonged, and both the surgeon and the patient require an unusual amount of fortitude and patience.

Meticulous care in the handling of tissues, exact approximation of skin surfaces, relief of tension and early removal of sutures are the primary requisites of scar prevention.

Depressed scars should be excised and a subcutaneous flap sutured below the skin before the new skin incisions are closed. Wolfe grafts for facial work are best handled by the technic that I described in the *Annals of Surgery* in 1927.

Congenital deformities, such as cleft lip, are among the more common facial disfigurements. When associated with cleft palate, cleft lip is one of the most distressing deformities with which a child may be affected. When the maxilla is involved, the operation on the bone should be done prior to the operation on the lip, as advocated by Brophy, in order to have the proper foundation for a normal lip and nose.

Probably in no other region does the plastic surgeon run the complete gamut of his armamentarium as he does in facial work, and even then his ingenuity is sometimes taxed to the limit to obtain a satisfactory cosmetic result, for every slight scar on the face is a potential disfigurement.

The question of cosmetic surgery per se resolves itself into a subject that sooner or later must be definitely faced by the medical profession. Ethical practitioners, alive to the irremedial havoc wrought by unlicensed and unqualified fakers, seek recourse in the education of a "none-too-willing" public, who are suspicious of the motives which activate their educational campaign while the quacks, quick to take advantage of the financial gain to be obtained, flourish and wax fat. While I have little patience with a surgeon who devotes his entire practice as a "sop to vanity," it must be remembered that vanity is a relative word and may be misconstrued. There are many cases in which the removal of age lines are legitimate both psychically and economically; if justified, this should be done by men who have had special training in this field.

The social and economic possibilities of the patient after cosmetic operations are sometimes astounding. All purely cosmetic operations should be done in a well organized hospital under strictly aseptic conditions. It is not an office procedure. The undercutting necessary is governed entirely by the extent of the disfigurement and must extend to the limit of the deformity. Stretching of the tissues must not be tolerated. Definite flaps must be made and raised, excess tissue removed and the freshened edges sutured to place with the least amount of scar in as inconspicuous a place as possible.

209 Post Street.

CANCER OF THE LARYNX

REPORT OF SEVERAL UNUSUAL CASES *

JOHN EDMUND MacKENTY, M.D.
NEW YORK

INTRINSIC BASAL CELL EPITHELIOMA OF THE LARYNX

CASE 1.—*History.*—K. H., a woman, aged 20, was referred by Dr. H. Edwin Lewis. The important points in the past history prior to subsequent development of cancer were: The patient was anemic and sickly as a child. She had a great deal of trouble during childhood with inflamed glands of the neck, and had many attacks of tonsillitis. She had had sinusitis since childhood. Hoarseness began in 1924 following acute laryngitis. It had been remittent ever since and had never been entirely absent. In November, 1926, the hoarseness deepened and became constant and progressive.

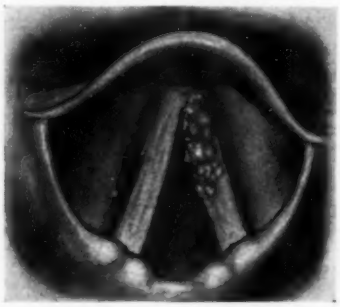


Fig. 1 (case 1).—Larynx at time of first examination, June 28, 1927.

Examination.—On June 29, 1927, examination revealed: chronic pansinusitis, badly infected tonsils, chronic laryngitis, neoplasm of the anterior half of the left vocal cord, and no change in mobility. Biopsy was done prior to my first examination. It was reported on by Dr. F. M. Jeffries, who stated: "In my opinion the growth on the vocal cord is papillary cancer. This type is slow growing and not prone to invade the lymph glands. It is often mistaken for sarcoma. It is undoubtedly malignant and I would advise radiation." At St. Luke's Hospital, in Dr. Francis Carter Wood's department, large doses of x-rays were applied for more than three months.

On Oct. 4, 1927, the whole larynx was tender, enlarged, rounded out and bulbous; the anatomic outlines were blurred; the skin was tanned and thickened; the subcutaneous tissues were adherent to the larynx. The larynx was filled with neoplasms on both sides, more on the left. The left arytenoid now seemed to be involved. The arytenoids were edematous. The breathing was obstructed. The patient was sent to Dr. Jackson to determine the condition of the hypopharynx and the esophageal mouth.

* Submitted for publication, June 4, 1929.

On Oct. 6, 1927, Dr. Jackson reported: "This is an acute edematous laryngitis and chondritis, with fungations in the larynx. The anterior wall of the hypopharynx is extremely inflammed and edematous, but the mucous membrane is still intact." The operation was delayed until Oct. 22, 1927, on account of the reaction following irradiation.

Operation.—On Oct. 22, 1927, with the patient under scopolamine-morphine, procaine-ether anesthesia, dense adhesions were found binding the larynx to its surroundings. The thyroid gland was large, covering two thirds of the larynx. There was a fibrous mass surrounding the larynx, suggestive of cancer. Frozen sections, taken at several points during the operation, revealed nothing of importance. The larynx, epiglottis and about one half of the thyroid gland were removed. The skin flaps were almost bloodless, and their immediate retraction

Fig. 2 (case 1).—Larynx removed, Oct. 22, 1927. Tremendous increase in growth in spite of intensive roentgen treatment.

at the time of operation made closure nearly impossible (the usual result after irradiation). The growth was found to involve both sides of the larynx, extending around the anterior commissure; its center was ulcerating and almost gangrenous, and its edges were raised into an indurated ridge.

The postoperative microscopic diagnosis was epithelioma, mostly of the basal cell type.

Convalescence was uneventful, and healing was slow. In thirty-three days the wound closed, with an unhealthy looking scar. Two weeks later, the scar broke down in the center, just under the chin. On December 27, the record stated: "Wound open and leaking and plastic operation seems inevitable." The wound was strapped, and the edges freshened by shaving with a sharp knife. My associates and I were afraid to perform a plastic operation and temporized. The wound finally closed, Jan. 5, 1928, two and one-half months after operation.

Comment.—It seems to me that as the years go by cancer is attacking younger and younger subjects. I am no longer surprised to find cancer of the larynx in patients in their thirties. This patient is the youngest seen. In spite of the apparent microscopic benignity of the growth, it grew with amazing rapidity, and clinically seemed to be highly malignant. Roentgen irradiation was useless; in fact, it not only nearly cost the patient her life in delaying the operation, but almost prevented the closure of the wound. The more I see of radium-burned tissue, the less I think of radium. A thorough irradiation of the neck for laryngeal cancer bears

Fig. 3 (case 1).—Appearance of patient, aged 20, showing final result.

almost a positive assurance that the wound in the neck (after operation) can never be closed. As far as I have observed, the x-rays are just as lethal to tissue as the radium ray.

Antecedent chronic laryngeal pathologic change was present also.

TUBERCULOSIS AND SQUAMOUS CELL CARCINOMA OF THE LARYNX

CASE 2.—*History.*—D. C., a man, aged 66 referred by Dr. Joseph Greene of Asheville, N. C., had had tuberculosis of the lungs for six years, with severe exacerbations. He had four pulmonary hemorrhages during this period. The x-rays showed extensive disease in both lungs, with formation of cavities. The sputum was always loaded with tubercle bacilli. He had had chronic parenchymatous nephritis and occasional glycosuria, with a blood sugar

content of from 180 to 190. The blood pressure was 172 systolic; 100 diastolic. Several years before admission, two operations were performed on the gallbladder, the last at the Mayo Clinic, for chololithiasis adhesions. The morphine and heroin habit was acquired during this period and had persisted ever since. When first seen, in June, 1927, the patient was taking about 4 grains (0.26 Gm.) of morphine a day to relieve the pain in the larynx, which was extreme.

At the Mayo Clinic, prior to the operation on the gallbladder a 2 plus Wassermann reaction was obtained; this was not confirmed elsewhere except that in Dr. Dunn's laboratory in Asheville spirochetes were said to have been found in secretion from a laryngeal pustule. In my opinion it is doubtful whether he ever had syphilis.

The patient was a liberal drinker of alcohol, though he was never known to show its effects, having an abnormally high alcoholic threshold.

Chronic hoarseness had been present for several years; the cause was unknown. In January, 1927, he developed acute laryngitis, with pain and tenderness over the left lateral wall of the larynx and dysphagia. These symptoms persisted until the time of operation (laryngectomy) in October, 1927. The pain was severe enough to require from 2 to 4 grains (0.13 to 0.26 Gm.) of morphine a day.

Examination.—My first examination was made on June 3, 1927. The patient was moderately hoarse and complained of constant laryngeal pain on the left side and of dysphagia. The left wall of the larynx was displaced inward by a deep-seated growth or inflammatory swelling. The mucous membrane of the larynx was congested, smooth and unbroken, except over the arytenoids where two grayish spots like blisters were seen, from which Dr. Greene had evacuated pus by puncture. The interarytenoid space and the right side of the larynx were normal except for congestion. There was complete fixation of the left side of the larynx and tenderness and some swelling over the external aspect of the thyroid cartilage on the left side. Here at one point, about as large as the end of the index finger, the tenderness was acute. At biopsy, on June 3, two good-sized pieces were taken from the area of greatest thickening just in front of the left arytenoid. Dr. Francis Carter Wood reported: "The laryngeal tissue shows only a chronic productive lesion of the connective tissue with large dilated blood vessels and highly cellular fibrous tissue. The diagnosis is doubtful. It might be well to give iodide of mercury. If no response, the lesion is probably tuberculous."

In July, 1927, Dr. Dunn of Asheville reported that in his laboratory *Spirochaeta pallida* was found in a small pustule in the patient's larynx.

Course of Illness.—I did not see the patient from June 3, 1927, until Sept. 27, 1927.

On Aug. 4, 1927, Dr. Greene reported: "The patient has been given six doses of neoarsphenamine. The growth seems to be reduced in size about one-third Unfortunately, he still requires morphine and codeine for pain. The swelling on the left side of the thyroid externally has almost disappeared, although there is still some tenderness which makes me fear chondritis or perichondritis."

On September 10, Dr. Greene again reported: "The arytenoid swelling has been greatly reduced although the swelling of the left vocal cord band is still very prominent. The pain is persistent. The tender spot on the neck is the same, but there is no swelling. I have punctured several pustules which were visible through the mucous membrane."

On September 27, I found the following condition: The laryngeal picture was quite different from that seen in June (my last examination). In the center of the laryngeal lesion there was a depression surrounded by a proliferating edge. From this crater-like opening a drop of pus was observed to exude, especially if

pressure was made over the tender area on the left outer wall of the thyroid cartilage. The growth or inflammatory mass was about the same size as it was in June. The inflammatory edema extended farther upward into the region of the aryepiglottic fold. From the appearance, malignant disease now seemed to be a certainty. The patient was expectorating daily about ½ pint (236 cc.) of tuberculous pus, looked fairly well and weighed 210 pounds (95.2 Kg.). The pain in the larynx was acute and distressing. A second biopsy was done by Jackson.

Dr. Wood's report on the examination of the tissue was as follows: "The sections show unquestionably squamous-cell epithelioma with undoubted tuberculosis in the

Fig. 4 (case 2).—Postero-anterior view of larynx. Cancer, tuberculosis and syphilis (?). The cancer has grown around the tuberculous fistula. See lateral view (fig. 5).

deeper layers. The epithelioma is rapidly growing, showing a large number of mitoses in the cells and is penetrating the deeper layers. The tuberculosis lies in the subepithelial layers. There are numerous typical tubercles with well-formed Langhans' giant cells, which is a totally different picture from the sections which I first saw and illustrates the difficulty of diagnosis."

Dr. James Ewing of the Department of Pathology of Cornell University, gave a more hopeful report. After stating that the growth was a "true epidermoid carcinoma of adult, squamous type," he went on to say that "the degree of cell anaplasia is slight and if one were to grade the case in order of malignancy one would class it as grade 1 among the true carcinomas. Therefore, we have to deal

with a squamous carcinoma of the larynx, papillary in type, of moderate malignancy and apparently of limited extent."

Operation.—On Oct. 10, 1927, total laryngectomy was performed, with the patient under scopolamine-morphine, procaine-ethylene anesthesia. The epiglottis, part of the mouth of the esophagus and a liberal section of tissue lateral to the aryepiglottic fold on the left side were removed with the larynx.

The complications that arose seemed endless and covered a period of seven months. Nine days after operation the patient developed a toxic delirium of pro-

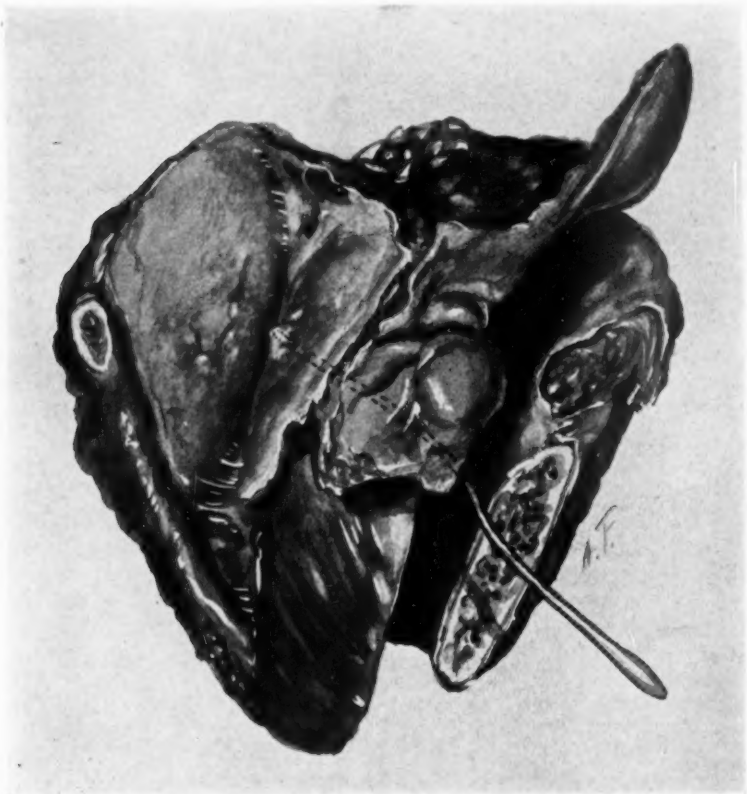


Fig. 5 (case 2).—Lateral view of larynx. Cancer, tuberculosis and syphilis (?) Tuberculosis involving perichondrium and cartilage. The probe is in the fistula. The cancer is in the lumen of the larynx around the fistula. Erosion on the thyroid cartilage should be noted (tuberculosis).

nounced degree. For two weeks restraint had to be used, except when he was under the influence of morphine. After this two weeks' active period, the mental condition was not entirely normal for several weeks. During this time the wound showed no signs of healing, a common result in postoperative toxic psychosis. The wound fell apart; the tracheal stump sloughed, and infection extended along the fascial planes of the neck in various directions. These pockets scarcely seemed to be inflammatory, since the surrounding parts showed none of the signs of a septic

inflammation. The fibrous planes of the neck melted, leaving finger-like extensions lined with a grayish slough. A curious phenomenon, and one never before observed, was that at no time during the healing process did a single granulation appear on the raw surface of the wound. The wound sloughed for several weeks, then turned a dirty gray, and slowly contracted until at the time this paper was written the condition shown in figure 6 was present. The skin became united to the mucous membrane along a slit opening into the esophagus about 1½ inches (3.7 cm.) in length. The tracheal opening appeared at the lower end of the slit and was separated from it by the esophageal wall. The rim of the trachea and the tracheal inner surface for ½ inch (1.2 cm.) down had never cicatrized. On them there had been constantly present irregular, very superficial, serpigenous ulcerations. These had a grayish base and were tender to the touch. I have taken them to be tuberculous ulcers. At times they would all but disappear, only to break out again, depending on the patient's general nutrition.

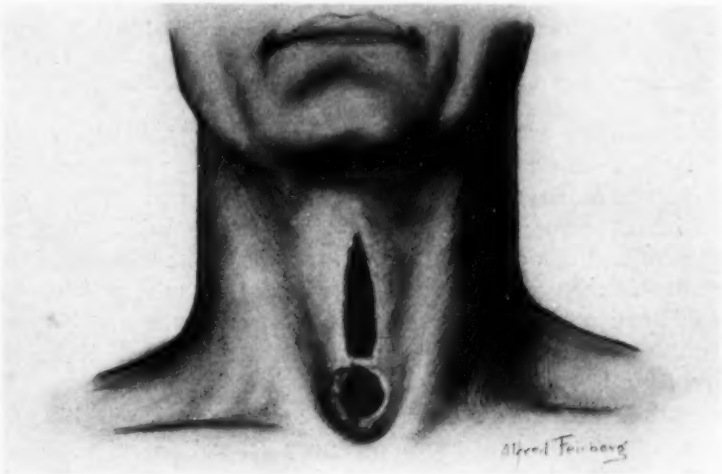


Fig. 6 (case 2).—Appearance of opening in esophagus and tracheal opening about May 1, seven months after operation. The raised edge on the right side of the tracheal opening and the ulceration on the posterior wall should be noted. This is a gradually healing, tuberculous process. Occasionally, there is an extension of the tuberculosis into the deeper tissues to the extent of an inch or more, forming sinus tracts which gradually heal.

Three weeks after the operation, hiccup appeared and in a few days increased to an alarming degree. Spasm of the cardia and regurgitation of most of the food taken complicated the hiccup. This spasm was so severe that the feeding tube at times could not be passed. This condition lasted about three weeks. At its maximum, the patient was at times unconscious and appeared profoundly toxic. These hiccups continued at intervals for five months, in two-week periods with about ten days, intervening.

During the periods of hiccup, the amount of urine would decrease to 7 or 8 ounces (207 to 236 cc.) in twenty-four hours. During the intervals, it would increase to 15 or 18 ounces (444 or 532 cc.). At last large daily doses of castor

oil were found to control the hiccup, which in the final opinion of the physicians was believed due to chronic toxemia.

The patient's weight decreased from 211 to 147 pounds (95.7 to 66.7 Kg.). On May 1, he weighed 158 pounds (71.7 Kg.) and was slowly gaining.

About six weeks after operation, the dosage of morphine was gradually reduced until 1/12 grain (5 mg.) was given hypodermically three times a day. The next step that had to be faced was total withdrawal or an inevitable increase of the drug. The patient was constantly fighting for more. A psychiatrist was called in, and after two weeks of strenuous misery, the habit was broken.

The blood sugar, which when treatment was not given ranged between 170 and 190, had to be controlled with insulin for weeks. It is now normal.

For months the output of urine was seldom over 15 ounces in twenty-four hours. It has now risen to 22 ounces (650 cc.). It still shows a pathologic condition of the kidneys.

On April 15, the patient had a pulmonary hemorrhage from a tuberculous cavity in the right apex and lost a quart of blood. He rapidly rallied from this under transfusion. Two years after operation, the wound had healed, except for the defect in the hypopharynx. The patient weighed 188 pounds (85.3 Kg.). There was no recurrence. The tuberculosis in the lungs is the same.

Comment.—Tuberculosis, cancer and perhaps syphilis were present in this larynx. An antecedent pathologic condition was present in the larynx for years, as shown by hoarseness. Pain and dysphagia were present from the onset, pointing strongly to the probability that tuberculosis antedated this cancer by several months. The tuberculosis probably started in the deep structure of the larynx near the perichondrium.

In this patient, just as in another in this series, the first biopsy was negative. The second was positive, but by that time the clinical evidence was unmistakable.

I cannot account for the extraordinary tissue reaction in this patient. It cannot be entirely explained by his poor general condition. His was the first wound I had ever seen which at no time showed a single granulation. Could the tuberculous sputum, constantly bathing it, account for this phenomenon?

The opening from the neck into the esophagus has proved a tremendous help in carrying out scientific feeding in this patient. All his life he has been a scant eater. His obstinacy is such that no persuasion could have forced his intake above 1,500 calories. On this amount he could not build himself up and at the same time fight the tuberculosis in the lungs. With the opening in the neck, it has been possible to give him from 2,500 to 3,000 calories daily.

A device, described later, placed in the esophagus, enables him to take food by mouth. It also conducts the saliva into the stomach, keeping it away from the tracheal opening. For want of a better name, I have called this an artificial esophagus.

The artificial esophagus is a device which, properly placed in the defect, directs the food and saliva toward the stomach. It is of inesti-

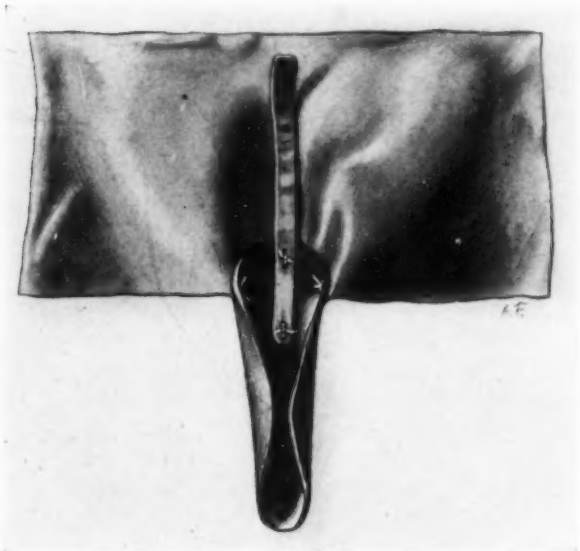


Fig. 7.—Artificial esophagus made of rubber tubing, rubber dam and block tin. When this is placed in the esophagus, the slit goes backward and the rounded upper end projects just about one-half inch below the upper angle of the opening in the neck.

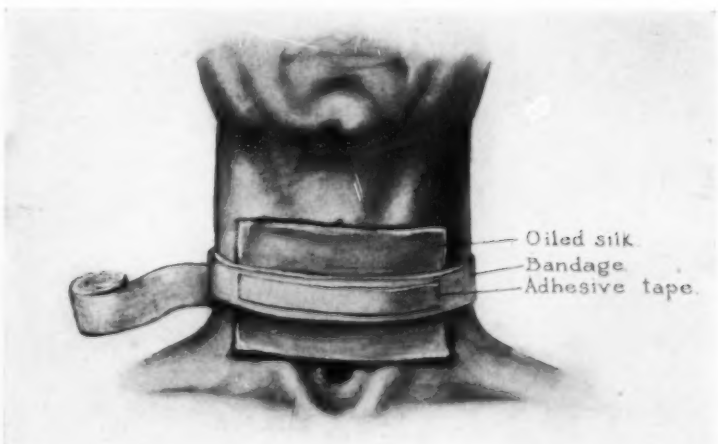


Fig. 8.—Method of applying the fixation point for retaining the artificial esophagus. The bandage is made of heavy cotton. About four turns, fortified with adhesive plaster at each turn, are used.

mable value in this case in keeping saliva out of the lungs, since on account of the incessant cough a tracheal cannula could not be used.

A specially shaped contrivance made of rubber tubing, rubber dam and block tin (fig. 7) is placed in the defect and is secured to a fixed point on the neck (figs. 8, 9, 10 and 11). The column of air which

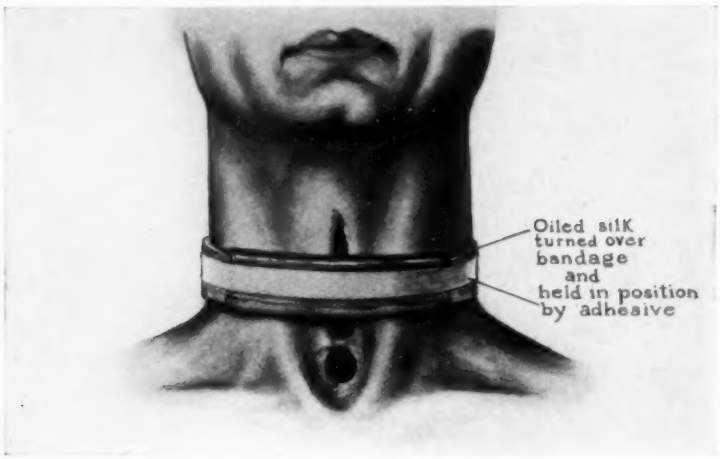


Fig. 9.—Fixation bandage completed: oiled silk, folded over and held in place by adhesive plaster. The object of the oiled silk is to keep the bandage dry.

Fig. 10.—Insertion of artificial esophagus into esophageal opening. It is pushed down until the upper rounded part of the apparatus lies behind the bandage. Then the rubber apron is spread out and tucked under the bandage and the block tin is bent acutely downward and fastened to the fixation point, or bandage. The upper part of this bend is grasped by the finger and thumb and bent slightly forward.

precedes each bolus of food escapes through the opening just above the rubber apparatus, allowing the food to fall without hindrance against its spoutlike projection and directing it into the lower part of the esophagus. The diverting of the air bolus, preceding the food, is the chief principle on which this device works, since it does away with air compression at the point of leakage.

By July 15, 1928, the patient had improved very much. His weight was 175 pounds (79.4 Kg.). The tuberculous process around the esophagus had practically healed. The amount of tubercle bacilli in the sputum had greatly decreased, and he was able to be about. On account of the tuberculous character of the infection around the trachea and the lower part of the esophageal opening, it seems to me that it will be difficult to do a plastic operation, since any traumatism in this tissue would

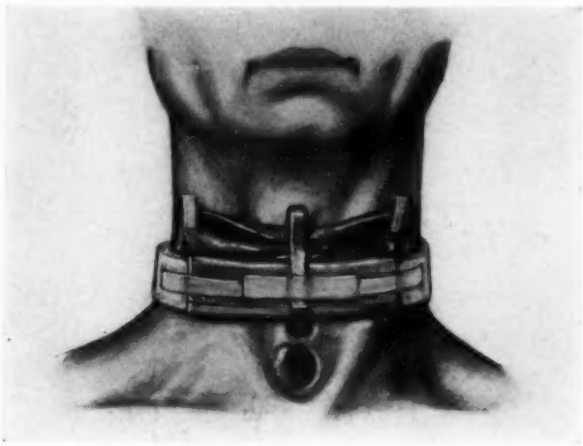


Fig. 11.—Apparatus in position. Note that the rubber apron is tucked under the bandage and fastened with adhesive tape to the neck; also that the block tin is turned down at an acute angle and fastened to the fixed point or bandage.

inevitably stir up a fresh attack of local tuberculosis. If a plastic procedure is attempted, it should be delayed for at least a year.

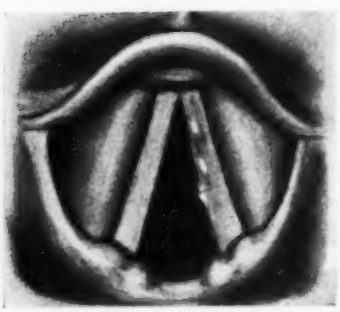
INTRINSIC SQUAMOUS CELL CARCINOMA OF THE LARYNX

CASE 3.—*History.*—E. G., a man, aged 67, referred by Dr. A. Richard Stern, was under observation for peculiar pathologic changes in the larynx from Oct. 14, 1922, until Dec. 27, 1927, when he was operated on for cancer.

The points in the past history bearing on the subsequent development of cancer were: For eight years prior to 1922, he had frequent attacks of coryza with hoarseness and at times aphonia. Six months before the first visit in 1922, the hoarseness became worse. The opinion of the consultants was that the hoarseness was caused by a greatly inflamed larynx. Dr. Jackson saw the patient in June, 1922, and said that the possibility of malignant disease could not be

eliminated without a biopsy. He advised a section. This was refused. Tuberculosis and syphilis were excluded.

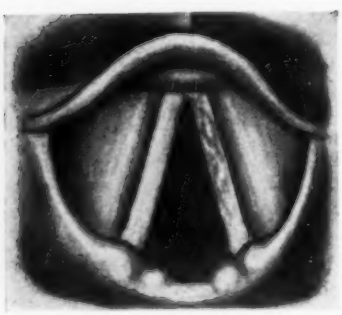
When I saw him in October, 1922, his voice was almost normal. The pathologic condition had affected only the left cord. There were small round spots on the upper surface of the cord (superficial ulcers); thickening on the edge at the middle and posterior thirds and congestion. The mouth was severely infected from pyorrheal teeth. Figures 12, 13, 14 and 15 show the changes that took place in the larynx up to November, 1924 (two years), at which date the larynx was normal. It remained so for two years, up to November, 1926. During this time he was taking intermittently small doses of sodium iodide, about 5 grains (0.3 Gm.) a day.



Oct. 14, 1922

Oct. 28, 1922

Nov. 11, 1922



Nov. 25, 1922

Jan. 24, 1923

March 3, 1923

Fig. 12 (case 3).—Drawings showing the peculiar changes in the larynx during the period indicated. The entire left cord seemed to be involved at various times, with a predilection for the junction of the middle and posterior thirds. The appearance was as represented, white, raised, recurring, keratotic areas.

In November, 1926, the hoarseness returned, and a white raised spot appeared on the upper surface of the left cord, about its center. I made the note that it looked like hyperkeratosis. From now to the end, the pathologic changes on the cord were single, not multiple, as in the earlier stages.

In March, 1927, the appearance was highly suggestive of incipient cancer, and the patient was sent to Dr. Jackson for an opinion. He agreed on the probability of malignant disease. A biopsy was advised, but the patient deferred it.

In April and May, 1927, the ulcer had shrunk to one-fourth its former size; it had filled in but had not closed. Bright red punctate points appeared in its center. The voice was better. In June the condition was worse, and a biopsy was insisted on. He was again sent to Dr. Jackson. The mobility was normal.

On June 25, 1927, Dr. Jackson reported: "I feel confident from the sensation transmitted to the forceps, as well as from the direct laryngoscopic appearance, that the growth is benign. Therefore, I deemed it best to remove the entire growth. My diagnosis is chronic inflammatory lesion."

One of my associates had the patient under observation from July 1 to August 6. He reported, on August 6: "The left cord is much improved. There is still a slight thickening. The mucous membrane looks quite normal."

Feb. 26, 1927 March 4, 1927 March 21, 1927

April 1, 1927 April 8, 1927 April 29, 1927

Fig. 13 (case 3).—Drawings showing the changes during the period indicated. The disease now seems to center entirely around the middle third of the cord.

Dr. Jackson saw the patient on August 20, and reported: "Taking the histological findings together with the appearance of mirror laryngoscopy the conditions in the larynx do not seem to me to call for any surgical procedure." The patient was not seen from August 20 to November 11.

About two and one-half months later, on November 11, the larynx presented the typical appearances of a fulminating cancer. The entire left cord was involved in a proliferating mass, and mobility was impaired. The patient was sent to Dr. Jackson for a second biopsy. The diagnosis was squamous cell cancer.

Because of a severe attack of grip, the patient was not in a condition to withstand operation until December 21. After all these years of watching and

circumventing every possible avenue of error, we now had, in spite of all our efforts, an advanced intrinsic cancer to deal with. If I had not had the unerring skill and judgment of Dr. Jackson covering every move, I might have blamed myself for overlooking the malignant condition in June. I am confident, however, that the actual malignant disease, that is, so far as our present clinical and laboratory experience can orient us, did not set in until after Dr. Jackson's last observation in August. Yet potentially, the condition was malignant long before.

Here is another example of the premalignant stage of cancer. I believe that this stage is invariably present, but opportunities for observing it come rarely.

Operation.—On Dec. 21, 1927, laryngectomy was performed. The growth extended close to the aryepiglottic fold. The left side of the larynx was entirely

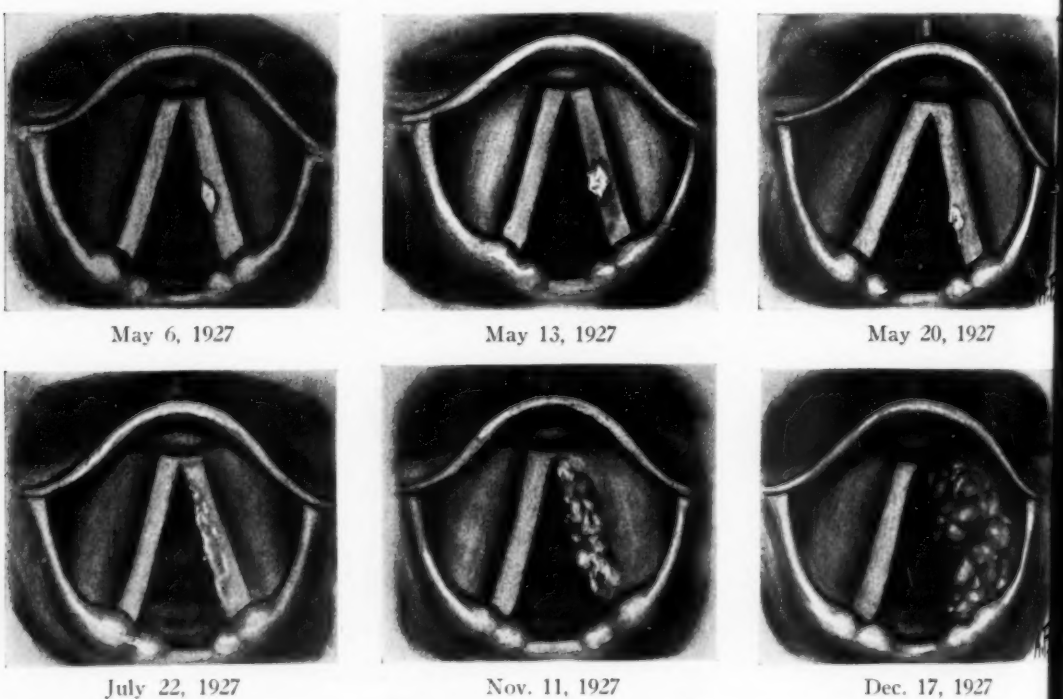


Fig. 14 (case 3).—Drawings showing the appearance of the cord during the time indicated. Biopsy done by Dr. Chevalier Jackson about June 25 was negative. On July 22, the cord seemed to be normal. The record shows that Dr. Jackson saw the cord on August 20, when it was normal. The tremendous rapidity of the growth of cancer between November and December should be noted.

involved. The tissues in the vicinity of the aryepiglottic fold on the lateral wall were extensively removed with the epiglottis. The recovery was complicated by osteoperiostitis (embolic) of the first right metatarsal bone. The temperature was 105 F. for several days. Eventually, the toe and part of the metatarsal bone had to be removed. In spite of this, the wound closed, and the patient was able to swallow fourteen days after the operation. The toe healed three months later. The patient died of cancer of the neck one year after operation.

Comment.—Under observation, an apparently benign growth became malignant. Or was the larynx cancerous from the start and the condition overlooked because of our abysmal ignorance of this disease? When more is known about cancer, will it be possible to differentiate between the truly benign growth never becoming malignant and the apparently benign growth always becoming malignant?

I felt for years that this larynx would some day present the typical picture of cancer. It is undeniable that certain appearances in the larynx suggest malignant disease to the experienced clinician long before the disease can be demonstrated clinically or biologically.

Fig. 15 (case 3).—Larynx after removal, showing extensive carcinoma growing downward and backward.

The rapidity of the growth was phenomenal, eventually defeating the painstaking efforts to stop it. Just a few weeks before the larynx was half filled with cancer, my associate and Dr. Jackson pronounced the condition practically normal. The first biopsy by Dr. Jackson was frankly negative.

The osteoperiostitis in the metatarsus was an embolic infection. Embolism and thrombosis, I believe, are complications of the operation of laryngectomy to be reckoned with. In old people, I have observed several cases of emboli in the brain causing slow death, as in dementia. In one case, the terminal stage was meningitis three months after operation. In my series this year, one case of phlebitis occurred, also a case of embolus into the radial artery, with recovery, in a man, aged 76.

SQUAMOUS CELL CARCINOMA OF THE LARYNX

CASE 4.—*History.*—W. C., a man, aged 60, referred by Dr. Harmon Smith, consulted me on June 15, 1925. Total laryngectomy was performed on Dec. 17, 1927, two years later. During the interval, the patient was under constant treatment with radium.

The points in the past history antedating the development of cancer were: The patient had syphilis thirty-five years before I saw him. During recent years, several examinations of the blood gave negative results. Hoarseness, varying in degree, had been present for the past five years; this was said to be due to papilloma of the left vocal cord. For a year before the patient consulted me, the hoarseness had been growing deeper and had been constant.

Examination.—In June, 1925, examination revealed a typical malignant growth involving three fourths of the left cord. There was partial fixation of the left cord and extension downward under the glottis. A biopsy report brought by the patient stated that the condition was squamous cell cancer. Total laryngectomy was advised but was refused.

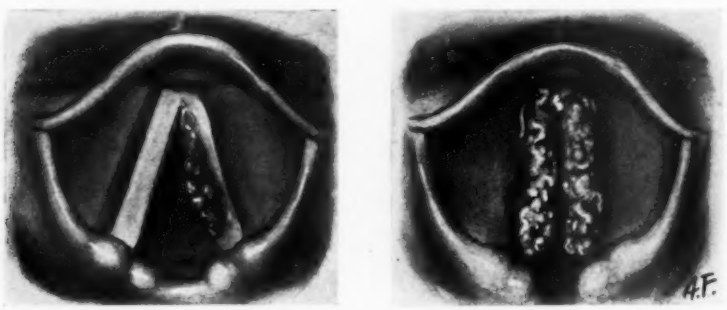


Fig. 16 (case 4).—Carcinoma of the larynx, showing growth extending over two years, at first intrinsic, later becoming extrinsic. The anterior angle of the thyroid cartilage was perforated by the disease.

The patient was next seen on Nov. 23, 1927, two years later. At that time, external examination revealed that the skin and subcutaneous tissues and the front and lateral aspects of the neck were severely damaged by radium. The skin was glazed, smooth, covered with minute blood vessels and tightly stretched around the neck. All tissue elasticity was destroyed. The larynx was an enlarged, rounded mass without anatomic outlines. There was softening of the cartilage over the front of the larynx.

Internal examination revealed that the larynx was filled with cancer; both sides seemingly were equally involved. There was complete double fixation of cords. The arytenoids were free from disease on their superior and posterior aspects. The hypopharynx was free. The patient was emaciated, weak and cachectic. Breathing was difficult and for weeks had prevented him from sleeping. He was taking scarcely any food.

Operation and Course.—Tracheotomy was done on Nov. 29, 1927. With the patient under local anesthesia, an incision 2 cm. long was made into the trachea and a tube inserted. It seemed advisable to traumatize this radium-burned tissue as little as possible, hence the small incision. The patient was then placed under the care of a metabolist for preparation for operation. He reported

FIG. 17 (Case 4).—Larynx after removal, showding bilateral involvement well forward. Perforation of cartilage at anterior angle.

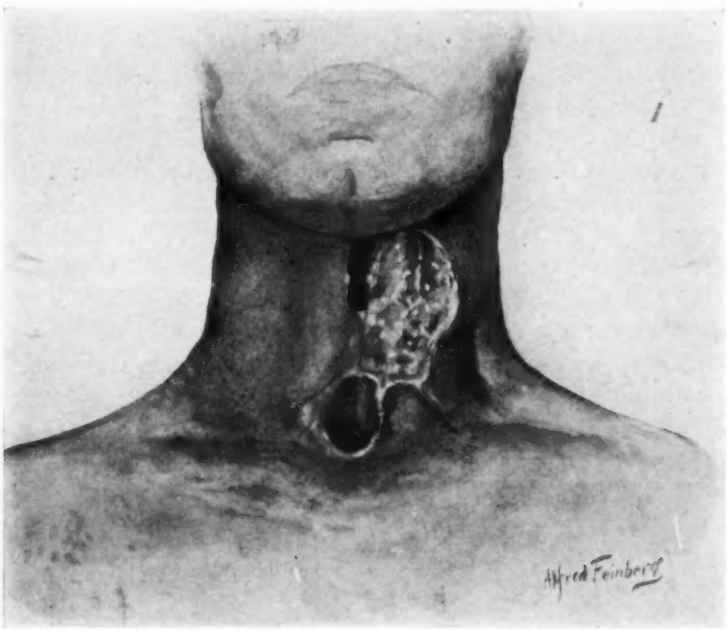


Fig. 18 (case 4).—Radium slough two months after the plastic operation failed to close the esophagus.

low blood pressure, a dilated aorta, emaciation from starvation, cancer toxemia, retention of urea nitrogen and uric acid, and kidney insufficiency. During two weeks of rest, forced feeding and metabolic correction the patient gained 15 pounds (6.8 Kg.); he then tolerated the laryngectomy normally.

On Dec. 17, 1927, the larynx, pretracheal muscles and skin, part of the thyroid gland and the tracheotomy opening with a short section of the trachea were removed *en masse*. All anatomic landmarks were obliterated, even the muscles being converted into fibrous tissue, by radium. When cut, the skin flaps receded like cut rubber and were practically bloodless. Dry gangrene attacked the wound at once, commencing at the edges of the cuts and extending outward until the whole front of the neck was involved. Then a mild erysipeloid skin infection began at the site of the wound and extended over the torso as far down as the waist.

On Feb. 8, 1928, the wound being clean and a large dehiscence being present in the esophagus, a plastic operation was attempted. The skin from the sides of the neck was brought forward. This operation was an utter failure; again gangrene removed the entire flap area. A 2 inch (5 cm.) defect remained in the front of the esophagus.

An artificial esophagus, described previously, was worn and enabled the patient to feed himself by mouth. This apparatus also conducted the saliva into the stomach and obviated the discomfort and irritation of saliva flowing over the neck. Death occurred from recurrence eight months later.

Comment.—It is possible that the irradiation retarded the growth of the cancer which, nevertheless, was slowly progressing to a fatal end.

The interior of the larynx, viewed macroscopically immediately after the operation, was almost gangrenous, probably owing to the shutting off of the blood supply by irradiation.

There was here a pronounced cachexia or toxemia, which disappeared rapidly after the offending mass was removed.

Treatment with radium converted this condition from an almost benign, easily curable disease in 1925 to a hopeless one in 1927. If cancer had failed to complete its cycle, the patient must have lived a more or less miserable and handicapped existence with an open esophagus.

Experiences like this make one feel that in patients who have received extensive irradiation the disease should be allowed to run its course and attempt should not be made to save them.

An antecedent chronic laryngeal pathologic condition was also present here, as in the other cases reported.

THE USE OF IODIZED OIL AS AN AID TO DIAGNOSIS IN SINUS CONDITIONS*

ARTHUR I. WEIL, M.D.
NEW ORLEANS

The last word in roentgenography of the sinuses has not been said. There is much that is unsatisfactory in the making and in the interpretation of x-ray plates in the diagnosis of sinus conditions. It is in the effort to throw a little added light on this subject and to give a somewhat new angle in its consideration that these studies have been made.[1]

It is true that the injection of an opaque material such as bismuth paste or molten metal into the sinuses of dried skulls and their roentgenologic study are old, and much of the knowledge of the interrelationship of the sinuses and their regional anatomy in the x-ray plate has been obtained by this means. Also there have been sporadic studies of the sinuses of living subjects by the injection of iodized poppy seed oil 40 per cent or similar opaque substances. What I am proposing, however, is a systematic and routine examination with iodized oil of such sinuses as give evidence of pathologic changes by the ordinary x-ray plate, but in which the roentgen observations are inconclusive, or inconsistent with the clinical picture.

I have found this method of investigation applicable to all the sinuses except the ethmoid, and, indeed, in some cases considerable light has been thrown on conditions in the posterior ethmoid region. The information obtained has been twofold: first, as to the character and extent of the pathologic changes present and second, the demonstration of anatomic variations from the normal, both in the size and in the location of certain sinuses which might confuse or give rise to incorrect diagnosis.

EXPERIMENTAL WORK

In each instance, injections have been made directly into the sinus investigated by means of a cannula passed into the cavity itself, the effort being made to inject that sinus alone, without getting iodized oil into the adjacent sinuses. The diffuse filling of all the sinuses in one region by means of the Proetz displacement and similar methods has

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* From the Otolaryngological and Roentgenological Departments of the Touro Infirmary.

1. In all of these studies, Dr. W. F. Henderson, the director of the X-ray Department of the Touro Infirmary, has taken an equal part with myself. He has directed and controlled all the roentgenologic portion of the study.

been purposely avoided; for it is my belief that for purposes of diagnosis, much more information can be obtained by filling the sinus thought to be infected and leaving the adjacent sinuses clear for contrast, than by filling all the sinuses indiscriminately. This is particularly true as regards the sphenoid and posterior ethmoid cells, the very region in which the diffuse method of filling has heretofore been advocated.

Dr. Henderson and I have made injections into a great many sinuses in the course of our work, and the figures shown are only a few selected to demonstrate some of the points made in this report.

Fig. 1.—Marked chronic hyperplastic right antrum. Note the very thick mucous membrane and the comparatively small area filled with iodized oil.

The antrum is the easiest sinus into which to make injections; indeed, there are practically no instances in which iodized oil cannot be introduced into this cavity. The injection is made through the ordinary Douglas cannula, introduced in the same manner as for washing the antrum. If pus is present, it is washed out first and the remaining water is removed by blowing air through the sinus, before the iodized oil is introduced.

It sometimes happens that when we try to wash out an antrum, even though we are sure that the cannula is in the antrum, no fluid

can be forced through. One such case is shown in this series of roentgenograms. There was no way of determining whether this obstruction was due to the presence of polyps filling the sinus, or possibly to the presence of a small polyp only in the region of the natural opening, blocking the return flow through that opening, or whether the antrum was filled with a large tumor mass. The iodized oil was injected with the Douglas cannula in place, and the subsequent roentgenograms showed that though the cannula was in the antrum, the only iodized oil visible was a few drops trickling back along the needle tract into the nose. A diagnosis was made of a tumor mass completely filling the antrum, which diagnosis was subsequently confirmed at opera-

Fig. 2.—Slight hyperplastic antrum. The iodized oil extends almost but not quite to the walls on all sides.

tion. After removal of the tumor, the antrum could be well filled with iodized oil.

In this connection, it might be mentioned that in making the control pictures after radical operation on the antrum, we experienced some difficulty at first in getting the pictures with the iodized oil in place. There was a tendency for the iodized oil, as soon as introduced, to escape through the large naso-antral opening left for drainage. Several methods were tried to avoid this difficulty, but the simplest and most effective was found to be to introduce the cannula into the antrum through this large opening, and then to pack wet gauze around the can-

nula in the opening and in the inferior meatus. After the iodized oil was injected, the cannula was removed, leaving the pack in place. This pack formed a temporary artificial wall to the antrum and retained the iodized oil in place long enough so that satisfactory pictures could be obtained.

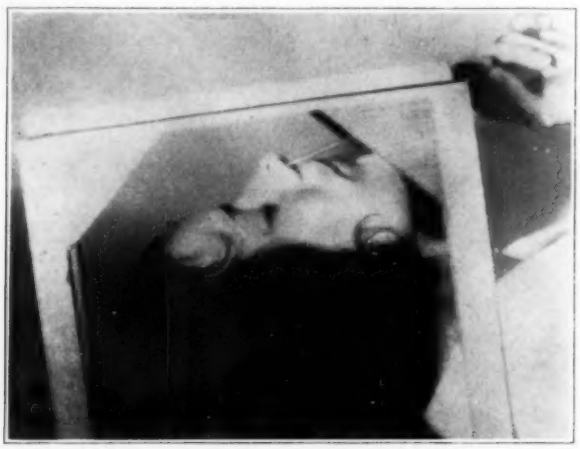

Fig. 3.—New position used in making injections of iodized oil into the frontal sinus. Note the cannula extending into the frontal sinus through which the iodized oil is introduced with the patient in correct position.

Fig. 4.—Marked hyperplastic condition of the frontal sinus. The iodized oil assumes the shape of the frontal but is held away from the walls by the thickened mucous membrane throughout.

A cloudy antrum in the x-ray plate usually means one of four things: an antrum filled with pus, one containing a large amount of polyps and swollen mucous membrane, a hyperplastic condition with its

accompanying thickened lining membrane or a tumor of the antrum. There is no way of differentiating by a simple x-ray plate between these four conditions. With the help of iodized oil, it is possible to distinguish between them and to determine the underlying pathologic cause. Examples of all four conditions are shown in our slides. Such differentiation is of great assistance in determining whether conservative or radical treatment is indicated.

We are often puzzled by an antrum which is dark to transillumination and to the x-rays, but in which, on washing, the fluid returns clear. The

Fig. 5.—Both sphenoids filled through the natural opening on the left side, showing dehiscence or absence of the septum between the sphenoids.

iodized oil makes possible the differentiation between tumors, hyperplasia or polyps as a cause.

What has been said of the antrum holds true to a large extent for the frontal sinus. Similar conditions exist there and a similar differential diagnosis can be made in those cases in which iodized oil can be introduced. It is not possible, however, as it is in the antrum, to inject the iodized oil in practically every case, certainly not without preliminary surgical procedure, such as middle turbinectomy. This we by no means advocate for mere diagnostic purpose, except in those cases in which we believe surgical intervention will ultimately be necessary

in order to cure the patient, the first step of which would be a middle turbinectomy. There is, however, a large percentage of cases in which the cannula can be introduced into the sinus without preliminary surgical intervention. Such patients are all subject to study with iodized oil. The cannula is put into place through the nasofrontal canal, and if pus is present, it is washed out and the sinus dried with air before the iodized oil is injected.

It was found, however, that the ordinary 23 degree angle for making the pictures was unsatisfactory for two reasons. It is impracticable to have the patient in the correct position with the cannula in place, and if the cannula is removed a large portion of the iodized oil may escape through the nasofrontal canal. Dr. Henderson accordingly devised a special position for making these pictures. The patient lies

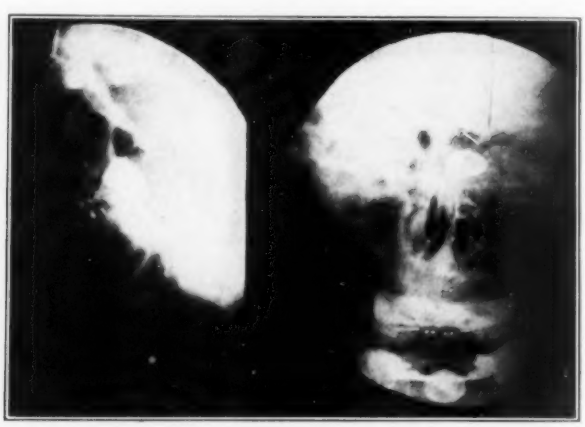

Fig. 6.—Large posterior ethmoid cell filled with iodized oil. There is no iodized oil in the sphenoid. Note how the large ethmoid cell extends as far back as the sella turcica, and the fallacy of attempting to measure the depth of the sphenoid with a graduate probe.

flat on his back on the fluoroscopic table with the cannula in place, the chin being raised to an angle of about 15 degrees. The x-ray tube is beneath the table, and the plate rests on a small platform raised on legs and inclined toward the head of the table at an angle of about 15 degrees. This platform rests just over the patient's face and practically parallel to it, so that the rays pass directly through the sinus to the plate. The iodized oil is injected into the sinus only after the patient is in place on the table. Figure 3 shows this position, and doubtless makes it more clear. With this position satisfactory, pictures of the frontal sinus have been obtained.

As has been stated, while this method of investigation has been of much service in studying the frontal sinus and antrum, its chief value, nevertheless, for several reasons has proved to be in the region of

the sphenoid. In the first place, it is in this region particularly that the simple roentgenogram is apt to be inconclusive, or inconsistent with the clinical observations; in the second place, it is here that anatomic variations from the normal are most apt to cloud or to confuse the diagnosis, and in the third place, the sphenoid is the sinus in which we are frequently least certain that the cannula has really entered that cavity. Indeed, in some instances, when we have felt the cannula pass definitely through a bony opening into a cavity of considerable size in the sphenoid region and when we have felt reasonably sure

Fig. 7.—The right sphenoid injected through the natural opening in the left nostril, showing how sphenoiditis on one side can cause pus in the opposite side.

that the cannula was in the sphenoid, the roentgenograms taken after the injection of iodized oil have shown definitely that the cannula was not in the sphenoid, but in a large posterior ethmoid cell. Some of these cells, as will be shown, are as large and as far back as a sphenoid. It is conceivable that in operating on a patient with such a case one might curet out a large posterior ethmoid cell and believe that one had cleaned out the sphenoid. Naturally, in such an event the operative observations might not coincide with the clinical diagnosis. This mistake is particularly possible when one is dealing with the sphenoid alone and not with the ethmoid as well. For if one has done a preliminary

middle turbinectomy and posterior ethmoidectomy, one is not so likely to be deceived as to entrance into the sphenoid.

When there is pathologic change in the sphenoid, it is likely to be either pus, polypoid degeneration, a chronic hyperplasia or possibly, in rare instances, a tumor. The ordinary roentgenogram shows cloudiness of the sphenoid in all these with variations in the Grainger line, but does not differentiate between them. We have roentgenograms taken after the injection of iodized oil which show all these conditions and how they may be distinguished one from the other. We have other

Fig. 8.—Probe in place in the nose resting against the anterior wall of the sphenoid, for roentgenologic orientation before attempting to enter that sinus with a sphenoid burr.

slides in which the preliminary roentgenogram showed the sphenoid equally opaque and the Grainger line equally thick, or indistinct, possibly due to variations in bone thickness or the like, but in which the iodized oil shows the sphenoid to be entirely free from pathologic change.

Grainger claims to be able to differentiate these conditions by a study of the ordinary x-ray plate. I shall not argue this point. It may be possible for a master mind, or one who has had the opportunity, as Grainger has, of studying thousands of roentgenograms of the sinus to make this distinction. I cannot say. But I do know that for most

physicians, without iodized oil, it is difficult, or well nigh impossible, to be sure of the underlying conditions. The iodized oil, however, clearly outlines the walls of the cavity, shows the thickness of the lining membrane, the presence of polyps or a possible tumor and makes the diagnosis much simpler.

In making roentgenograms with iodized oil in the sphenoid we have used the Grainger position, that is, the 107 degree angle, very largely and have found it satisfactory. In addition, we usually make a side exposure to give a lateral outline of the sinus. We study both positions in order to reach a diagnosis.

Fig. 9.—A normal sphenoid with iodized oil extending to the smooth walls on all sides. The preliminary x-ray plate without iodized oil showed marked clouding suggesting sphenoid disease.

In addition to the pathologic changes, we have demonstrated certain anatomic variations. Though we have always known that such variations occur in certain persons, we have heretofore had no means of determining in which particular patient they are present. This is important, for such variations can sometimes give rise to an error in diagnosis.

In one roentgenogram the injection of iodized oil into the right sphenoid fills the whole sphenoid cavity, right and left, due probably to an absence of, or a dehiscence in, the septum between the two sides. In such a case sphenoiditis attributed to one side might easily cause ocular or other symptoms on the opposite side. In other roentgenograms the sphenoid on one side is so much larger than on the other that it encroaches materially on the other side, extending well beyond

the median line. We have had one or two cases in which the sphenoid is so enormous that it practically fills the other side, leaving little space for the sphenoid of that side. Such a condition might give rise to a double error. In the first place, in operating, say on the left side, it would be easy to enter the right sphenoid and not touch the left sphenoid. In the second place, such a large sphenoid extending far to the opposite side could easily cause ocular or other nerve symptoms on the left, when it was really the right sphenoid which was at fault.

A third anatomic peculiarity is the presence of those large enclosed posterior ethmoid cells mentioned as large as a sphenoid cavity and

Fig. 10.—A large sphenoid extending well to the opposite side. This sphenoid is otherwise normal though the preliminary x-ray plate showed evidence of sphenoid disease.

situated as far back in the nose. Several of these roentgenograms are shown. Such a cell might possibly be mistaken for the sphenoid in operations on this region. Moreover, these cells are of especial interest in showing how easily one might be deceived if the attempt is made, as has been recommended by Dupuy and others, to determine whether the sphenoid has been entered by measuring its depth from the nasal spine with a graduated probe. One case is shown in particular in which the large posterior ethmoid cell extends posteriorly to the depth of the sella turcica and, if judged by depth alone, one would feel certain that the cannula was in the sphenoid. We are forced to the conclusion, by these observations, that such method of orientation is faulty.

In two or three of our cases, in which injections have been made into the sphenoid, on one side through its natural opening, we have been surprised to find that it was the sphenoid of the opposite side which was injected, while that on the same side remained empty. This would seem to show that the sphenoid does not always open into its own side of the nose and that pus coming from the sphenoid region on one side of the nose does not necessarily mean that it is the sphenoid on that side which is infected.

Another interesting fact has been disclosed. We have noticed that whereas the frontal sinus and antrum empty themselves of the iodized oil rather quickly, so that a plate made a day or two after the injections finds these cavities free from iodized oil, the same has not always been

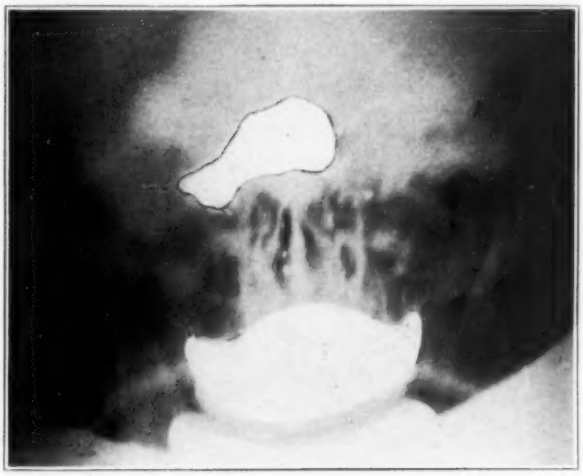

Fig. 11.—An enormous right sphenoid not only filling almost all the left side also, but likewise extending so far into the wing of the sphenoid that it is doubtful if the ordinary nasal sphenoid operation would reach this area.

true of the sphenoid. We have found that it occasionally retains the iodized oil for a long time, and in some instances plates made as long as a week or ten days after the injection show the sphenoid to be as full as when it was first injected. Irrigation of the sphenoid at this time shows the presence of a large quantity of iodized oil. This would seem to indicate that the sphenoid is not nearly so readily drained as the other cavities, which would mean probably that the sphenoid does not recover so quickly as the other cavities from acute purulent infections. It might also explain the relative frequency of chronic hyperplasia of that region.

The criticism has been made by some with whom this study has been discussed that this method requires too much surgical intervention

and that it is not altogether devoid of danger. This is emphatically not the case. We use no surgical measures in introducing the iodized oil, except in the case of the antrum in which it is done by means of a Douglas cannula. Injections are made into the other sinuses through their natural openings. Only in those cases in which we believe from the preliminary x-ray plate that surgical intervention will ultimately be necessary is any patient subjected to surgical procedures. In the case of the frontal sinus a middle turbinectomy might be needed, though up to the present time we have not found that necessary in any of our

Fig. 12.—Filling defect in both sphenoids. The preliminary x-ray picture showed normal sphenoids but on account of this filling defect and persistent unexplained eye disturbance double sphenoidectomy was performed with observations which confirmed the diagnosis of hyperplastic sphenoiditis.

cases. There have been a few sphenoids, however, in which it was impossible to pass the cannula through the natural opening and in which we thought from the roentgenogram that an operation on the sphenoid would be necessary. In some of these cases the anterior wall of the sphenoid has been penetrated with a sphenoid burr and the cannula introduced through this opening. This is done, however, only under roentgen control, as follows: A probe is introduced into the nose to the anterior wall of the sphenoid; with this probe in place a roent-

genogram is made showing just where it rests against the anterior wall and that the cavity is sufficiently large to make entrance with a burr perfectly safe. Only then is the burr introduced.

This systematic study of the sinuses after injections of iodized oil is still new and not enough work has been done with it as yet to outline definitely all its fields of applicability and its limitations. We cannot afford, therefore, to be too dogmatic in our statements and conclusions. It is only in the hope of encouraging others to similar lines of investigation and of provoking some discussion, either favorable or otherwise, which may throw some new light on the subject, that our results up to the present time are being reported.

CONCLUSIONS

1. The injection of iodized oil helps frequently in diagnosing and treating sinus conditions in which the ordinary roentgen observations are inconclusive or uncertain.

2. Injections are made into the sinus alone, leaving the adjacent sinuses clear for contrast.

3. Various pathologic conditions, such as simple hyperplasia, polyp formation or tumors, can be differentiated by this method.

4. Certain anatomic variations can be demonstrated which might give rise to incorrect or uncertain diagnosis.

5. No surgical intervention, other than puncture of the antrum, is used in introducing the iodized oil, except in certain selected cases and then usually only under roentgen control.

NOTE.—Since this article was submitted for publication, my attention has been called to a paper by Dr. Hilding Anderson of Duluth, Minn., on the same subject appearing in the ARCHIVES (**7**:340 and 514 [April-May] 1928), which has heretofore inexplicably escaped my notice. His technic, x-ray plates and conclusions are so strikingly similar to mine that in justice to myself I wish to state that my studies were made entirely without knowledge of his work. Had I known of his paper I should have been glad to have given him due credit.

319 Physicians and Surgeons Building.

SIMPLIFIED INVESTIGATION OF THE SPHENOIDAL SINUS *

M. C. MYERSON, M.D.
NEW YORK

The sphenoidal sinus is frequently disregarded or overlooked in the routine investigation of disease of the nasal sinuses, and it is almost always overlooked as a possible source of origin of severe general infections. This may be because some otolaryngologists feel that actual infection of the sphenoidal sinus is of relatively infrequent occurrence. Probably less than 5 per cent of all the sphenoidal cavities that I have opened or have seen opened have shown the presence of active infection. Fraser [1] has recently stated that a great many unnecessary operations are being performed on this sinus. In his experience, a small percentage of these sinuses have been found involved at the time of operation. For this reason, it would seem proper that investigations of the sphenoidal sinus should be confined to such cases as present signs and symptoms which may be directly referable to the sphenoid.

Broadly speaking, there are two groups of cases that require such investigation. The first group comprises such cases as manifest local symptoms which could be directly traced to the sphenoidal sinus. Here one would consider a symptom complex that would include any or all of the following: pain in the back of the eyes, pain over the mastoid region, occipital and vertical headache, postnasal discharge, enlargement of the blind spot, mental or physical apathy, loss of memory, nausea and vomiting. In the second group would be patients suffering from general sepsis or meningitis of undetermined origin. Several years ago, Skillern [2] stressed the importance of rhinologic examination in all cases of meningitis of doubtful origin.

There are many isolated case reports which tell the story of a latent sphenoidal infection relieved by operation or disclosed at autopsy. The symptoms of latent disease of the sphenoidal sinus vary. Jonathan Wright [3] recorded a case of isolated unilateral latent empyema of the sphenoidal sinus accompanied by delirium and mental symptoms which

* Submitted for publication, Oct. 14, 1929.

1. Fraser, J. S.: Frequency of Sphenoidal Sinus Operations, J. Laryngol. & Otol. **39**:393 (July) 1924.

2. Skillern, R. H.: The Importance of Rhinologic Examination in All Cases of Meningitis of Doubtful Origin, Penn. M. J., August, 1909.

3. Wright, Jonathan: A Case of Unilateral Empyema of the Sphenoidal Sinus with Delirium and Mental Symptoms Relieved by Operation, Ann. Otol. Rhin. & Laryng., February, 1902, p. 17.

were relieved by operation. Grayson's [4] patient complained of mental hebetude, recurring headache and impaired nutrition. Hubert's [5] patient complained of lack of concentration, recurring stupor and mental and physical apathy. The principal symptoms of Stout's [6] patient were disturbance of vision, severe headache, dizziness, nausea and stiffness of the joints and muscles. Lyman's [7] patient suffered from a simulated mastoiditis with severe pain and tenderness in the region of the left ear. Ridpath [8] reported a series of cases demonstrating the presence of infection of the sphenoidal sinus and the variety of symptoms which may be caused by this infection. Bertemes' [9] patient suffered with occipitomastoid pain and wryneck for fourteen years.[10]

Patients suffering with general sepsis or with meningitis in which the spinal fluid is still free from bacteria are of special interest. Faulkner [11] reported the case of a woman who suffered with chronic sepsis for several months and then suddenly developed signs of meningeal irritation. Opening of the sphenoidal sinus caused her recovery. Toubert's [12] patient suffered a fatal meningitis which was found to be due to an acute sphenoiditis at autopsy. It is such cases as these which should have the benefit of an investigation of the sphenoidal sinuses.

The simplest form of investigation of the sphenoidal sinus is the sounding of this cavity. This may be accomplished with the aid of direct vision in a wide nasal chamber or by the sense of touch when the ostium cannot be brought into view. The passage of a sound through the ostium could be accomplished in 40 per cent of noses, according to

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4. Grayson, C. P.: The Exploratory Opening of the Sphenoidal Sinus, Laryngoscope **25**:65 (Jan.) 1915.

5. Hubert, L.: Sphenoidal Sinusitis with Marked Cerebral Symptoms; Operation, Recovery, Laryngoscope **31**:322 (May) 1921.

6. Stout, P. S.: An Interesting History of a Patient with Sphenoidal Sinus Disease, Laryngoscope **34**:379 (May) 1924.

7. Lyman, H. W.: Simulated Mastoiditis Relieved by Sphenoidectomy, Laryngoscope **34**:948 (Dec.) 1924.

8. Ridpath, R. F.: Sphenoidal Sinusitis with Peculiar and Unique Symptoms, Laryngoscope **37**:880 (Dec.) 1927.

9. Bertemes: Neuralgie vidienne dans le cours d'un sinusiteé sphenoidale chronique, Ann. d. mal. de l'oreille, du larynx **33**:735, 1924.

10. The many and varied symptoms of disease of the sphenoidal sinus are given by Skillern (The Accessory Sinuses of the Nose, ed. 4, Philadelphia, J. B. Lippincott Co., 1923, p. 381) and by Ridpath (footnote 8).

11. Faulkner, E. R.: Sphenoidal Sinusitis with Marked Cerebral Symptoms: Operation; Recovery, Laryngoscope **31**:323 (May) 1921.

12. Toubert, F.: Acute Sphenoidal Sinusitis Terminating in Fatal Suppurative Meningitis and Diagnosticated Only at Autopsy, Med. Bull., October, 1900.

Holmes [13] (1896). The other minor surgical or conservative procedure for the investigation of this sinus is puncture of the anterior wall. This is done with one of the several trocars available for this purpose. This procedure necessitates either forceful dilatation of the olfactory sulcus to separate the middle turbinate farther from the septum or the removal of the posterior end of the middle turbinate.

Those who have performed operations on the sphenoidal sinus or have had the opportunity for work on the cadaver must have been impressed with the extreme thinness of the anterior wall of the sphenoidal sinus. This has been demonstrated in the work of Zuckerkandl,[14] Onodi,[15] Mosher,[16] Loeb,[17] W. B. Davis,[18] Schaeffer [19] and, more recently, Neivert.[20] Early in my experience with sphenoidal operations, I was impressed with this observation. I soon came to effect entrance into the sphenoidal sinus by means of pressure on the anterior wall with the tip of a silver nasal probe. Because of this frequent thinness, it has occurred to me that the employment of heavy instruments such as trocars was perhaps not necessary. I have therefore been employing an ordinary silver nasal probe for the preliminary puncture of the sphenoidal sinus.[20a] After the probe has entered the sphenoidal cavity, a blunt ended cannula of a similar thickness is introduced.

TECHNIC

My experience has shown that the recumbent position is to be preferred. If it is not expedient to have the patient in the recumbent position, the sitting position may be used. When the patient is lying down, the head is tilted backward so that the posterior wall of the sphenoidal sinus lies in a horizontal plane. The olfactory sulcus is now cocainized with four or five applicators wound in cotton saturated with 10 or 20 per cent solution of cocaine with epinephrine hydrochloride. Each succeeding applicator is insinuated more deeply toward the face of the sphenoidal sinus. The last application is made to this surface. It is important to

13. Holmes, C. R.: The Sphenoidal Cavity and Its Relation to the Eye, Arch. Ophth. **25**:460, 1896.

14. Zuckerkandl, E.: Normale und pathologische Anatomie der Nasenhöhle und ihrer pneumatischen Anhange, Vienna, 1882, vol. 1.

15. Onodi, A.: Die Nebenhöhlen der Nase, Vienna, Alfred Holder, 1905.

16. Mosher, H. P.: Anatomy of the Sphenoidal Sinus and the Method of Approaching It from the Antrum, Laryngoscope **13**:177 (March) 1903. This article contains an extensive bibliography.

17. Loeb, H. W.: The Cubical Capacity and Superficial Area of the Sphenoidal Sinus, Ann. Otol. Rhin. & Laryng. **21**:1, 1912; ibid., June, 1909.

18. Davis, W. B.: Nasal Accessory Sinuses in Man, Philadelphia, W. B. Saunders & Company, 1914, p. 160.

19. Schaeffer, J. P.: The Nose and Olfactory Organs, Philadelphia, P. Blakiston's Son & Company, 1920, chapter 5, p. 175.

20. Neivert, H.: Morphological Variations as a Factor in Symptomatology of Paranasal Sinus Disease, Arch. Otolaryng. **1**:367 (April) 1925.

20a. Roentgenographic study of the sphenoidal sinus should precede its puncture in all cases.

note during the last application what is the exact distance of the anterior wall of the sphenoidal sinus from the tip of the nose, as measured by the applicator. For this distance must be noted and used when the probe is introduced. This anesthetization usually takes about five minutes. The application of the solution serves to shrink the mucous membrane of the septum and middle turbinate where they may be in contact, and the end of the applicator wound with cotton serves as a wedge to create the space required for the instrumentation. When necessary, the shaft of the applicator is swung outward so as to separate these structures further.

After cocainization, the olfactory sulcus and the face of the sphenoid are swabbed with alcohol. A silver nasal probe the end of which is bent at an angle of 170 degrees about 1 inch from the end is passed along the channel which has been prepared for it. A mark has previously been made on the probe to correspond with the length of the applicator which was required to reach the anterior wall of the sphenoidal sinus. When this wall is reached the handle of the probe is tilted upward so that the shaft is more or less horizontal (sitting position). The probe is now manipulated so as to attempt to enter the ostium of the sinus. It has been my experience that with the expenditure of sufficient time one can enter the ostium of a large percentage of sphenoidal sinuses in this way, surely 75 per cent. However, if the natural orifice is not entered, the probe is so directed that the tip points slightly downward. Firm pressure is applied at the handle, and the anterior wall is readily pierced. If the probe is directed too high, pressure directed on it will cause the probe to bend on itself. This is due to the malleability of the probe and to the resistance of the roof of the nose which is not readily penetrable. When the anterior wall is penetrated, one can feel the sudden passage of the probe into the sinus cavity. The sensation imparted when this occurs is unmistakable. In some cases the degree of angulation may have to be changed because of a septal deviation posteriorly. At times it may be necessary to bend the end of the probe in two different planes.

After the probe has penetrated into the sphenoidal sinus, it is withdrawn and its angulation is noted. A special silver cannula with blunt end is similarly angulated and is passed along the same route that the probe traversed. This cannula is of approximately the same thickness as the probe and has no stylet. The cannula is readily passed into the sinus in this manner. A 5 or 10 cc. syringe is now attached to the proximal end of the cannula, and the cavity is aspirated. If nothing is obtained by aspiration 2 cc. of sterile saline solution is introduced into the sinus and withdrawn back into the syringe. If this fluid is purulent or cloudy, it is transferred to a tube and sent to the laboratory for study. Strict asepsis must be observed in this procedure.

DIFFICULTIES AND DANGERS

It is impossible to follow this technic when there is a marked angulation or convexity of the septum which will not permit a view of the olfactory sulcus.

Bleeding will occur if the operator is not gentle in his manipulations. Bleeding occurred in the experience of Hinkle,[21] Hajek [21] and Gleitsmann. In Myles' [21] case the bleeding was so severe that it was necessary to ligate the external carotid artery. In this instance, trephining

21. Quoted by Gleitsmann, J. W.: The Sphenoidal Sinus, Laryngoscope **15:** 621 (Aug.) 1905.

and curettage were resorted to. Bleeding should not occur when a bulbous, tipped silver probe is used in the first step of this procedure. Gleitsmann mentioned that a patient of Scholz suffered a fatal hemorrhage as a result of a perforation of the cavernous sinus.

I have noticed as a result of studies on the skull of the cadaver that it is comparatively easy to pass a probe through the sella turcica if one is not careful. This should be avoided by measuring the distance to the face of the sphenoid and noting this when the probe passes through the anterior wall, and by the angle at which the probe is introduced.

Schaeffer called attention to the fact that occasionally the posterior wall of the sphenoidal sinus is quite thin. In such a case the posterior wall might be penetrated when undue or unnecessarily prolonged pressure was used. The roof of the nose is more liable to injury when a

Fig. 1.—Nasal probe used to puncture anterior sphenoidal wall.

Fig. 2.—Special cannula for entering sphenoidal sinus after puncture has been made with the probe.

firm, pointed instrument is used than when a dull ended nonresistant one is employed.

The contention that the cribriform plate or roof of the nasal cavity in the region of the spheno-ethmoid recess might be injured by the instrumentation which I have described will not be substantiated by experience. Several years ago, I had the opportunity to study the sinuses and their adjacent structures in a large number of cadavers. At that time I was impressed with the relative thickness of the cribriform plate and of the roof of the posterior portion of the olfactory sulcus. It was noticed that they could not be penetrated unless a good deal of force was used on a sharp or firm instrument. In this connection, the following is quoted from Skillern: [22]

22. Skillern (footnote 10, p. 391).

Much has been said of penetrating the cribriform plate during this maneuver (sounding the sphenoidal sinus with a probe). This is more apparent than real, for that structure lies too far forward and the posterior portion of the roof of the olfactory fissure is quite thick at its juncture with the sphenoidal sinus and does not yield readily to an instrument as delicate as a probe. Indeed I have intentionally endeavored to puncture the plate on numerous cadavers but have failed in every instance, the sound bending before infraction of the bone was accomplished.

A great deal of work has been done on the sphenoidal sinus since 1882 when Hyrtl made the statement that the sphenoidal sinus was

Fig. 3.—Nasal probe in sphenoidal sinus.

beyond the reach of human attack. The investigation of the sphenoidal sinus by means of exploratory puncture is not new. As a result of his pioneering investigations of the sinuses, Zuckerkandl first offered the suggestion that the sphenoidal sinus could be penetrated by means of a trocar. This suggestion was first successfully followed by Schaeffer.[21] In 1894, Laurent [23] reported his method of sounding the sphenoidal sinus by means of an instrument shaped somewhat like a eustachian

23. Laurent: Catheterization and Exploration of the Sphenoidal Sinus Without a Speculum, J. Laryng. **8**:821, 1894.

catheter. For the past thirty years or more, P. Watson Williams [24] has been investigating the sphenoidal sinus by means of a firm cannula through which he introduced a trocar to pierce the anterior wall. In recent years, the trocar has been replaced by a blunt-ended stylet. After the trocar pierces the sphenoidal wall, the cannula is advanced into the cavity and the puncturing part of the instrument is withdrawn. P. and E. Watson Williams have been the most active advocates of this type of investigation of the sphenoidal sinus and have had a great deal of experience in this work. Sieur and Jacobs [25] brought forth a cannula shaped and curved like a eustachian catheter which they contended would follow a groove in the roof of the nasal cavity which directed the cannula into the ostium of the sphenoid. The studies of Mosher [16] showed that this was true in about 50 per cent of the dried specimens that he worked with. Menzel [26] has been using a special puncture needle which is bent at its end at an angle of 120 degrees. Hageman [27] also used a special trocar for entering the sphenoidal cavity. Lobell [28] has suggested a trocar with a cross piece so situated that it will prevent the passage of the instrument too far into the sphenoidal sinus. Spielberg [29] has also recorded his preference for a trocar which is essentially the same in plan and principle as the original Watson Williams instrument.

Fletcher Ingals [30] and Zeim [30] advocated entrance of the sphenoidal sinus through its floor by approach through the oral cavity by means of a right-angled drill.

Those who favor the use of a trocar or a heavy styletted cannula require that the nasal chamber shall be well anesthetized so as to permit the forceful separation of the septum and the middle turbinate to provide the necessary space for the passage of these instruments. If this is not sufficient, they advocate the removal of either the posterior portion or all of the middle turbinate. In a majority of noses all that is necessary for the passage of the silver probe or special cannula is the shrinkage incidental to the cocainization. This is the same technic that Skillern

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24. Williams, P. Watson: Rhinology, Lonodn, Longmans, Green & Company, 1910, p. 244.

25. Sieur and Jacobs: Recherches anatomiques, cliniques et operatives sur les fosses nasales et leur sinuses, Paris, 1901, p. 282.

26. Menzel, R. M.: Ztschr. f. Hals-, nasen- u. Ohrenh. **8**:185 (May) 1924.

27. Hageman, J. A.: Sphenoidal Sinus Trocar, Eye, Ear, Nose and Throat Month. **3**:206 (May) 1924.

28. Lobell, A.: Puncture Irrigation of the Sphenoidal Sinus with a New Instrument, Laryngoscope **36**:270 (April) 1926.

29. Spielberg, W.: A New Sphenoidal Trocar and Cannula, Laryngoscope **38**:122 (Feb.) 1928.

30. Quoted by Holmes (footnote 13).

advocates for the production of anesthesia preliminary to the sounding of the sphenoidal sinus with a probe.

A survey of the various instruments that are in use or are advocated will suffice to impress on one the relative safety and utility of the method which I advocate. The cannula that I employ is of solid silver and is malleable, so that it bends when it meets with much resistance. At the proximal end of the cannula there is a thumb piece which is similar to that found on the nasal probes. The purpose of the thumb piece is to acquaint the operator with the relative position of the distal end of the cannula when that end has been especially angulated for passage through the olfactory sulcus and into the sphenoidal sinus.

LIMITED LESIONS OF THE BASILAR MEMBRANE*

EDMUND PRINCE FOWLER, M.D.
NEW YORK

If the hearing sensitivity is plotted graphically, the line joining the points of minimum audibility will seldom, if ever, be an even line, but will vary at certain frequencies up and down. This is the case in both normal and abnormal ears. By the use of tuning forks, cylinders, whistles and sirens, it was discovered that in certain abnormal ears, so-called "tone-gaps" occurred; in other words, that within certain narrow frequency ranges, no sound was heard by the ear being tested, although the frequencies to each side of these areas were easily heard.

Audiometric measurements [1] prove that the true tone-gap is not a fact, because one never finds a true tone-gap. The reason no sound was formerly heard in these so-called tone-gaps was that the sounds used for testing were not loud enough. If the sounds had been loud enough, they would have been heard. Thousands of audiograms have been studied, and it can still be stated that no true tone-gap has been found.

The perfected 1*A* audiometer, with attachments for producing the frequencies lying between the octaves, half and third octaves, which the instrument normally sounds, enables one to explore at any frequency from below minimum audibility to maximum audibility, the latter being a sound so loud that much increase in intensity would cause pain. Although no true tone-gap has ever been found, with this instrument there have been discovered in several apparently normal ears and in many abnormal ears, marked deafened areas which are so deficient in hearing that the older instruments would not have detected any hearing in them. These areas are regularly contained within the upper frequency range (above 1,000), and are frequently limited to small areas near 4,000 double vibrations. It has been stated by others that one possible defect of the audiometer was its inability to detect deafness if it lay between the frequencies produced by the instrument, namely, between octaves, half or third octaves. This is not the case, because the narrowest deafened area (tone-gap) yet reported covered five-sixths of an octave.[1] The 1*A* audiometer, therefore, would have detected deafness even in this narrow area without the special attachment used to guard against such a fate. Even the 2*A* audiometer would have indicated marked deafness in all but a few instances. The frequency

* Submitted for publication, Aug. 13, 1929.

1. Fowler, E. P.: Marked Deafened Areas in Normal Ears, Arch. Otolaryng. 8:151 (Aug.) 1928.

intervals were originally planned to guard against the possibility of a gap, and they appear to have served the purpose admirably.

In the graphs shown, sufficient exploration was made between the usual frequencies of the 1*A* audiometer, to prevent serious error, but it is a time-consuming process to test many intermediate points and nothing important will be gained by doing so in routine testing.

Before the American Otological Society, in 1928, there was reported a case with a marked deafened area in an apparently otherwise normal ear.[1] Since then, repeated observations compel the conclusion that this was not a normal ear, because from time to time, concurrently with nasal inflammations there appeared distinct congestion in the attic always with some depression of the hearing, as shown by the audiometer, although little or none was complained of by the patient. Moreover, twenty-four striking examples of the condition have been accumulated, including not one normal ear, although many subjects thought that their hearing was perfect.

It is questionable just what limits should be established within which a limited deafened area may be said to be narrow, because opinions differ on this point. A reasonable definition would appear to be that a narrow marked deafened area is one in which the curve of hearing is sharply depressed, at least 20 sensation units for a distance of three octaves or less, or in which the sharp depression occurs near or above the frequency area of 4,000, whether or not it returns toward normal at a higher frequency. This definition will suffice for present purposes.

OBSERVATIONS

In analyzing these cases, it will be of advantage to first set down the following observations:

1. The dip occurred with nerve deafness, with obstructive deafness and with both. The greatest depressions always occurred in the upper frequencies (above 1,024).

2. The area of 4,000 double vibrations was regularly favored.

3. The hearing for the lower tones was often normal or near normal. In one instance it was 60 sensation units down.

4. Hearing for the tones above the dip was sometimes normal, but usually was half way down, and often little or no better than at the bottom of the dip.

5. The form of the curve varied in depth, width and the level of its brinks.

6. There was little variation in the regularity of the slopes of the curve.

7. The depth of the depressions varied from 20 to 100 sensation units. Unless 20 sensation units down by air conduction and bone conduction, they cannot be deemed of definite significance.

8. The width of the depressions varied from five sixths of an octave to three octaves (seldom over two).

9. The low tone side of the brink of the dip varied from normal to 50 sensation units down, and the high tone side from normal to below the capacity of the audiometer.

10. In several instances, previous audiograms showed the absence of any narrow dip, but in none of these was the hearing near normal in the upper frequencies.

11. In every instance, bone conduction (at dip frequencies) was markedly diminished, often in like amount to the air conduction.

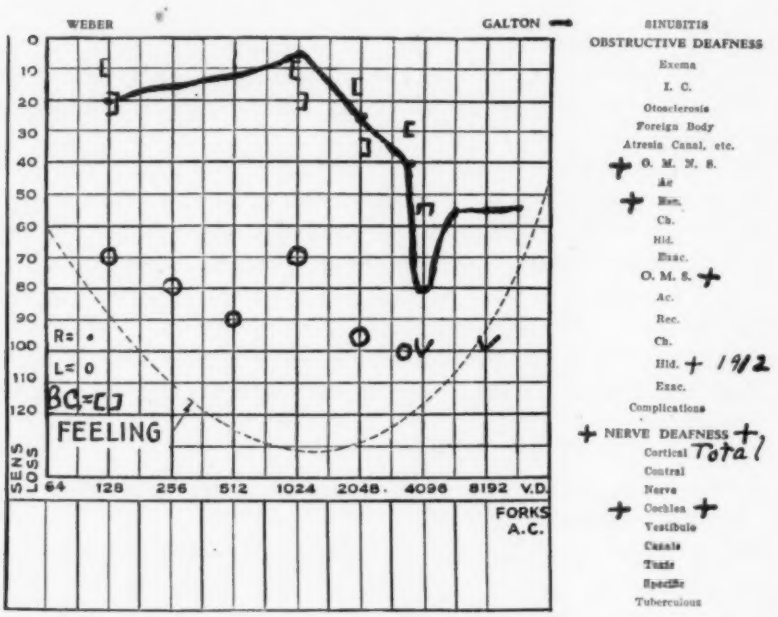

Chart 1.—Francis R. B., aged 31, heard normally after a mastoid operation in 1912. He was deafened for twelve hours on the left side following the explosion of an ammunition dump in France, in 1918. He did not realize for some time that total deafness was present in the left ear. Hearing in the right ear was normal for the voice, and nil in the left ear. The diagnosis was nerve deafness in both ears, total deafness in the left ear. The bone conduction follows the air conduction curve.

12. Whenever the bottom of the dip subsequently receded toward normal by air conduction, there was also improvement in the bone conduction.

13. In no instance did air conduction or bone conduction subsequently recede completely to normal at dip frequency.

14. Both dip brinks may subsequently improve to the same extent as the bottom of the dip.

15. In but few ears was there much improvement in the brink on the high tone side of the dip.

16. Tinnitus was regularly present (in about 66 per cent), and sometimes accentuated the depression of hearing within the dip frequency.

17. The tone of the tinnitus regularly corresponded to the dip frequencies, usually to a frequency on the low tone side of the slope near the greatest deficiency point.

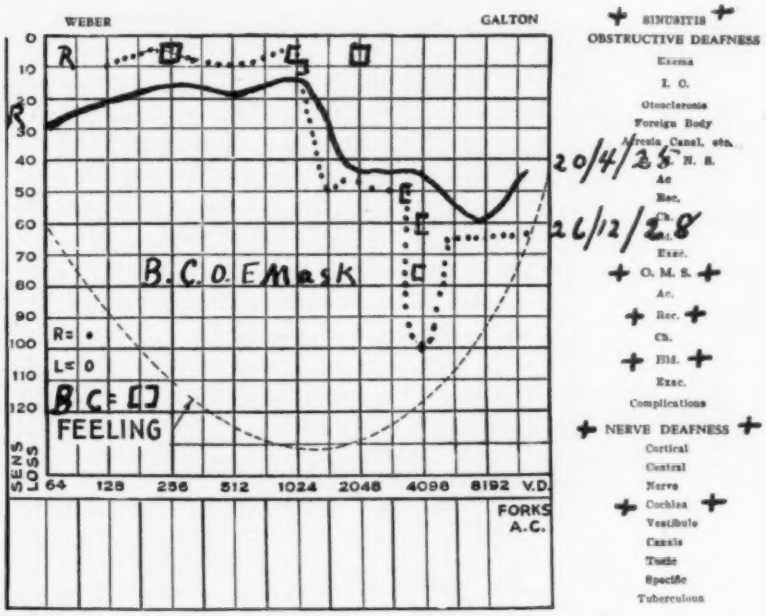

Chart 2.—R. C. W., aged 29, following an explosion of gas in 1910 was very deaf in both ears for two days. He had been somewhat deafened ever since, especially in the right ear, the condition of which is shown in this graph. He was subject to chronic nasal sinusitis with polyps. He had done much shotgun firing. The brink of the dip at 3,000 double vibrations did not change more than 5 sensation units, but in three months both the air and bone conduction at 4,000 improved 25 sensation units. Tinnitus was absent during this improvement. For several months it remained absent. His left ear has a marked narrow dip between 3,000 and 5,000 double vibrations, the bottom of the dip being at 55 sensation units, and the brinks at 10 sensation units. The graph shows only the curves for the right ear.

18. Whenever tinnitus disappeared or lessened, the dip was lessened from 5 to 15 sensation units, depending on the prior intensity of the tinnitus.

19. The greatest intensity of any tinnitus was estimated at 10 sensation units above minimum audibility. Hence, tinnitus can account at most for a loss of from 10 to 15 sensation units by masking.

20. Attempts to produce the phenomenon of beats with the tinnitus was unsuccessful. One notable exception to this observation is known.

21. Disease of the nasal sinus was present in every case, often, though by no means constantly, worse on the side of the deafer ear. The dip was not eliminated, though it improved considerably in some cases with relief from the sinusitis.

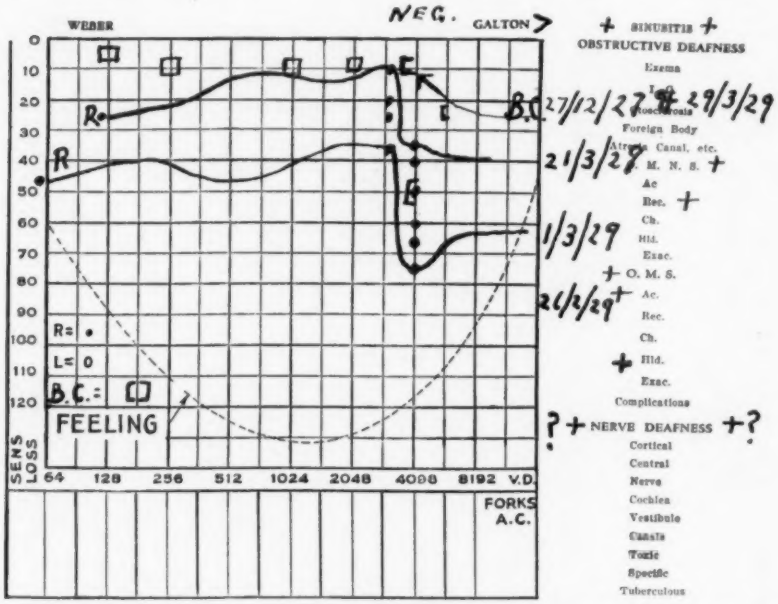

Chart 3.—H. O. B., aged 52, had chronic hypoplastic sinusitis in the right antrum with edema of the ethmoid septum on both sides. There was an increased shadow from inflammation of a healed mastoid antrum on the right side. Congestion was seen along the malleus handle of the right ear. The diagnosis was acute otitis media suppurativa on the right side which subsided with imporvement in the hearing at all frequencies including the dip frequencies (in which the bone conduction returned to normal) to approximately the same degree, as if the whole plane of hearing had been bodily lifted about 30 sensation units by air conduction. The bone conduction at the lower frequencies did not change. There was considerable tinnitus present at first, but this disappeared when the hearing had improved to 40 sensation units at 4,000 double vibrations, and did not return thereafter.

22. Politzerization in no instance brought about any improvement in the dip frequency. This fact eliminated obstructive lesions of the middle ear as a cause of the dip.

23. In seventeen instances there was definite history of acoustic trauma; i. e., trauma caused by gun fire, violent explosions, etc.

24. In several instances toxic factors were suspected.

25. Children showed dips only occasionally (four cases were in children).

26. When one ear was normal, masking it caused a greater loss by bone conduction in the opposite deafened ear at dip frequency, but masking the affected ear occasioned little or no loss by air conduction or by bone conduction in the normal ear at dip frequency. This was probably because the deafness at dip frequency was so great that the noise of masking lacked intensity sufficient to cause much masking.

COMMENT

Of course, until some of these ears have been serially sectioned the exact lesions causing limited deafened areas must remain uncertain, but by indirect methods an attempt to solve the problem is justifiable and may not lead one far astray.

It is evident that any lesion producing a narrow area of tone deficiency must lie somewhere along either the nerve mechanism or the sound conduction mechanism of the ear. In spite of the theoretical possibility of antiresonance filtering out certain frequencies, and changes in tension, weighting and friction depressing certain tones,[1] the narrowness, depth and characteristics (often appearing within the same range of tone) and of these dips, and the observations on bone conduction make it quite certain that the cause does not lie in the conduction mechanism of the ear. Moreover, if the conduction mechanism was the determining factor, it is almost inconceivable that the areas of deafness would be very narrow or that bone conduction would always be much below normal. The nervous mechanism of hearing would seem to offer the best field for search, but as explained in an earlier paper,[1] lesions in the cortex, centers and auditory nerve do not stand the test of close analysis. Therefore, the cochlea alone remains, and in the cochlea the end organ and the basilar membrane appear most promising, because the peri-endolymph, by no stretch of the imagination, could be so altered as to transmit exceedingly well all the frequencies except the few lying within the narrow dip areas. It is conceivable, however, that acoustic or mechanical shocks through the peri-endolymph channels might traumatize certain limited areas more than others.

It is interesting to trace the course and effects of trauma from a loud sound (for example, from a gun or cannon shot or other explosion) within the ear: 1. The explosive force will travel through the external auditory canal to the drum membrane. 2. Thence it will proceed through the air in the middle ear to the external labyrinth wall

and the oval and the round windows. The latter, being more movable than the stapes (5:1) and having no joint cushions or inertia due to attached ossicles, will prephase the stapes in its inward excursion. 3. As the ossicles are especially adapted for transmitting force to the stapes they will, as usual, greatly increase the force of the explosion from the membrana tympani to the stapes. 4. Through the perilymph and endolymph (acting as one) by hydraulic pressure, all contents of the cochlea will be compressed. 5. If the pressure is everywhere equal and opposed, it will cause no trauma unless there is some resultant motion. 6. Motion of the labyrinth walls cannot occur unless they rupture. Motion of the round window membrane will occur, especially on its outward rebound, and, reinforced by the impulse through the stapes, will force the basilar membrane into the scala tympani along its entire length, i. e., similar to the reception of normal sound waves, only in exaggerated form. 7. The basilar membrane, being narrower at its proximal end and wider distally, naturally has more play distally, and hence more motion can take place distally without injury, such as tearing or dislocation of the tissues. The narrower (high frequency) end is more delicate than the broader (low frequency) end, and is more prone to suffer from mechanical, chemical and nutritional insults. 8. The first whirl of the cochlea in man is constricted about 10 mm. from the proximal end, and this constriction will (1) cause an eddy in the explosive force, a rebound toward the basilar membrane between the 7 to 10 mm. point, and (2) force a venturimeter effect, that is the effect produced whenever a fluid is forced through a constriction between two spaces.

The fluid in the cochlea, suddenly compressed, is forced through the constricted area with violence, but the effect is the reverse of what might be first imagined. There is not an increase in the pressure at this constricted point, but a definite and strong negative pressure. (A small loss in pressure also occurs from friction and eddies as the flow progresses up the cochlea.)

Here there are, then, operating side by side within a narrow area, a violent positive pressure and a strong negative pressure. A sheering stress must occur which the delicate structures on the basilar membrane cannot withstand without distortion and dislocation (severe trauma in any case). Everything tends to concentrate in this narrow area. If the shock is not too violent, only a small limited area may be damaged. If great enough, the whole of Corti's organ may be damaged or destroyed.

I am including examples of mild and of violent acoustic shocks, cases showing both little damage and total destruction. Severe shock occurred in seventeen cases. It cannot be disproved in others, but for

the sake of argument, I am assuming that the dip might be occasioned otherwise. By what other means would this be possible?

The cochlea artery branches opposite the 7 mm. frequency area (4,000 double vibrations) and this may account for weakness in some ears (a deafened spot).

Infection and inflammation beginning in the middle ear might extend through blood and lymph channels into the labyrinth and cause congestion, edema or exudate, especially of the immediately underlying structures, such as the endosteum and stria vascularis, etc., but how these could skip the highest frequencies and attack a limited area just below them is unknown.

Pressure on the auditory nerve might affect principally the outer fibers which conduct the higher tones, but pressure would usually affect all or a large number of the outer fibers alike, and constant tinnitus at dip frequency would not be caused by pressure on the nerve alone. The picture in my typical cases suggests trauma as the cause of these very narrow areas of tone deficiency, and in every typical case a limited nerve deafness, as evidenced by a depression in both air and bone conduction. Supporation of the middle ear in one case apparently accentuated the depression; on healing, the dip returned almost to its status before the infection. It is easily conceivable that in such cases any prior nerve deafness might be accentuated, as this regularly occurs in all suppurations of the middle ear superimposed on nerve deafness.

If tinnitus is present, this also will depress the hearing. There was a marked tinnitus present in the case mentioned, and the improvement shown at dip frequency was about 15 sensation units greater than the improvement at all the other frequencies. The tinnitus would account for the difference of 15 sensation units because of the masking effect shown at the dip frequency during its presence. Tinnitus may occur with reactions of the sympathetic nerve causing vasoconstruction from angiospasm, or vasodilatation from relaxation, of the cochlear branches of the auditory artery, but deafness at many frequencies results, not deafness at a small part of the scale. Moreover, angiospasm is intermittent, and the dip deafness in these cases was not. Localized congestion, edema, hemorrhage, emboli, thrombi, etc., appear capable of causing limited lesions of the basilar membrane, but these may and do occur with severe acoustic trauma.

Several attempts to produce acoustic trauma in animals have failed from unfortunate accidents, but further efforts have been made, and soon the temporal bones will be ready for sectioning and study.

Toxic neuritis should be mentioned as a possible factor in any nervous lesion. Toxic factors cause deafness, especially at the higher frequencies and thus may accentuate the dip both vertically and laterally, the latter to such an extent that the upper half of the dip does not show

on the graph, but except for the theory that they attack best the point of least resistance, one cannot see how toxic factors alone can be held accountable for any of the typical depressions observed.

In a few instances (all acute inflammatory conditions), the most deafened portion of the deafened area subsequently receded to its pre-inflammatory status. It may be that in these cases a transient toxic neuritis affecting the weakest portion of the cochlea (the area of 4,000 double vibrations) was the cause of this transient accentuation of the dip.

Thus far, no marked narrow deafened areas (loops) have been noted in otosclerosis, possibly, because the otosclerotic ankylosis of the stapes protects the cochlea from acoustic trauma.

SUMMARY

Limited areas of more or less marked deafness may occur with nerve deafness, obstructive deafness or both. The area of 4,000 vibrations is regularly favored. Bone conduction at dip frequency is regularly lowered, and if not, one should suspect error in the determination of air conduction and carefully check up the test to insure accuracy. Unless a dip is 20 sensation units down, it cannot be deemed of any importance, even if bone conduction is to some extent lowered. Variations of this kind and the exact meaning of observations on bone conduction near 4,000 vibrations is problematic. Tinnitus is regularly present and may accentuate the depression of hearing within the dip frequencies.

It would appear that the medium high frequencies were particularly sensitive to various insults, especially to acoustic trauma and toxic neuritis, because they regularly fail to escape in these conditions, and so-called nerve deafness does not occur without deafened areas in this region. It would appear that in many ears there is an element of toxic neuritis or trauma diagnosable by the presence of a marked deafened area (dip) and otherwise unascertainable.

By not testing air conduction at several high frequencies (between 1,000 and 11,000) otologists are missing information of importance. Bone conduction should also be taken at at least one low frequency and at 1,000, 2,000, 3,000 and 4,000 vibrations, because by the common fault of omitting the latter frequencies many errors in diagnosis occur.

Small limited lesions in or on the basilar membrane appear sufficient to account for the phenomenon of "marked narrow deafened areas" in the human ear.

114 East Fifty-Fourth Street.

Clinical Notes

EPISTAXIS OF ANTRAL ORIGIN

Report of Cases *

G. B. Trible, M.D., Washington, D. C.

Various authors state that from 90 to 99 per cent of all bleeding from the nose is due to a local vascular defect, and that 90 per cent of this is situated in the anterior portion of the septal cartilage in Kiesselbach's area. This area was first described by an American named Little. The vessel in question is a small branch of the internal sphenopalatine, and bleeding usually occurs at about its anastomosis with a branch of the superior coronary, or artery of the septum, where there is a little racemose group or minute plexus, superficially situated.

The conditions affecting this large majority are multiple; there are local causes, such as traumas, ulcers and engorgement of all sorts, and the general causes, such as high blood pressure, altered states of the blood, as for instance the anemias and leukemias, puerpura, alteration of the coagulability, vicarious menstruation, changes in atmospheric pressure, administration of drugs, and to those who go back to the typhoid era, it can be recalled that the onset of typhoid was frequently ushered in by an epistaxis. Later, influenza has had the same rôle.

The sinuses, and particularly the antrum, have not been considered, except as a site for bleeding from malignant growths, or bleeding mucofibromas. It is only when discovery of no local site or no constitutional condition can be found that attention must be paid to other sites, and the bleeding from the area under the middle turbinate must be included. This should not be confused with the bleeding from the anterior ethmoidal vein, which occurs between the middle turbinate and the septum.

The possibility of epistaxis of antral origin, other than from new growth of extreme vascularity, does not loom up ordinarily as a factor in the mental sifting of causative factors in the making of a diagnosis.

That it exists and is a factor was first brought home to me in the first case to be reported.

* Report of Cases

Case 1.—Capt. P. F., U. S. N., reported that an extremely severe nose bleed had occurred about ten days subsequent to a submucous septum resection, presumably from the incision. Bearing in mind the possibility of a postoperative perforation, which may occur from devitalization or trauma, days and even months after an uncomplicated resection, careful search was made over the septum, but no trace of bleeding or blood was found, the edges of the wound uniting by first intention.

The patient was instructed to avoid too much exercise or heat, was given some palliative treatment and told to report again in a few days. That same night he reported the occurrence of another hemorrhage, and this also was reported by the medical officer in the Yard. On examination, again nothing was found, not even a clot.

A day or so later, the patient was sent from the Yard Dispensary, with free nasal bleeding from the right side, while the septal incision was in the left. No

* Submitted for publication, Sept. 6, 1929.

* Read before the Northern Virginia, Southern Maryland and District of Columbia Medical Society, May, 1929.

bleeding point could be found in the septum, but after great difficulty, owing to the profuse bleeding, it was found that the blood came from under the right middle turbinate. The right side was packed, with small strips of gauze, and the mouth was examined. The patient was asked whether his teeth were all right, to which he replied that had had some pain in the right upper second molar, and on investigation, the soft tissue around the tooth was swollen and painful.

Dr. Sterling Mead, exodontist, was called to look at the tooth, and he pronounced it diseased and advised its removal. There had been a history and evidence of sinus disease of long standing; so the procedure determined on was extraction of the tooth, and if connected with the antrum, to open the antrum after the Luc-Caldwell method. After extraction, a large wound was left in the antrum, fresh blood and older clots were found, and on opening the antrum, a bleeding point was found back of the area of the perforation, apparently from an erosion into the nutrient artery.

Case 2.—M. X., a woman, aged about 50, gave a history of nose bleeding for several days, in spite of repeated packing. Removal of packing slowly revealed no bleeding point in the septum, nor in the turbinates.

Transillumination showed cloudiness to the right, and a history was elicited of an old root, left from a previous extraction. No further bleeding occurred. The patient was sent for dental treatment, and the trouble has not recurred. Some swelling of the tissue below the eye led to the location of the bleeding side in this case.

Case 3.—Mrs. X. complained of profuse bleeding from the nose, in spite of packing, following dental extraction. No opening into the antrum could be found, but probably the trauma had broken into the artery along the floor. Cocainization, puncture and washing the antrum cleaned out fresh blood and clots, and no further filling occurred. It is not unlikely that bleeding in the antrum may frequently occur and cease from pressure of clots, and be noted only when there is an exceptionally patent ostium, or accessory ostia.

The following two cases, seen by Dr. William A. Morgan, and given to me to report, illustrate: the first, the bleeding from a malignant growth or the secondary anemia resulting from them and the second, that from what is probably arteriosclerotic changes, such as come in advanced life.

In the first of these the possibility of a developing metastasis must be borne in mind.

Case 4.—A white boy, aged 12, complained of bleeding from the right nostril, which had recurred from time to time for the past six months. There was a history of a malignant condition of the liver (sarcoma?) which had been diagnosed by bioscopy at operation, and apparently successfully treated by x-rays. Hemoglobin was between 60 and 70, and the general appearance was anemic. The broken eroded areas were cauterized, bleeding was checked, and the patient was advised to return if further troubled. Some time afterward, he returned with free bleeding from the left nostril, which was found coming from beneath the left inferior turbinate. This was checked by packing, but that night he had severe pain on the left side of the face and his teeth on that side ached. Transillumination showed an opaque left antrum, and after cocainization the antrum was punctured and washed out, and an almost solid clot was removed, which was dark and chocolate covered. The pain subsided almost immediately but the hemorrhage kept up; so he was hospitalized and given a transfusion. Other punctures and irrigations were made for a few days to keep down pressure pain in the antrum, and he made a slow recovery and has not had further trouble for the past two months.

CASE 5.—A woman, aged 64, gave a history of severe hemorrhages lasting for several hours which had to be checked by postnasal tampons. On examination, the right nostril was found filled with a large clot, extending from the middle turbinate to the postnasal space. On its removal, oozing was noted from under the middle turbinate. Transillumination showed a cloudy right antrum, and pressure elicited tenderness. Puncture and washing brought out a large clot, sufficient to fill the cavity of the antrum. Relief from pain was instantaneous, but the next day the antrum had refilled. After the second washing, which likewise removed a large clot, there was no recurrence.

The exact vessel or location of the bleeding in these cases cannot be definitely ascertained, unless there are other indications for opening the antrum.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

INTRADURAL CONDITIONS IN RELATION TO RHINOLOGY AND OTOLOGY*

WELLS P. EAGLETON, M.D.
NEWARK, N. J.

I. ADVANCES IN THE KNOWLEDGE OF THE ANATO-PHYSIOLOGY OF NERVOUS TISSUE IN RELATION TO THE NOSE AND EAR

This year's otologic literature is characterized by the large number of the cases reported and the high grade of the literary style employed and by the lack of new facts or ideas. It is chiefly in the domain of anatomy, physiology and neuropathology that real progress is apparent.

"NEUROBIOTAXIS (A PHYSICAL LAW IN THE STRUCTURE OF THE NERVOUS SYSTEM)"[1]

The structures in the central nervous system are arranged according to the impulses that pass through them. Proximity or junction of central nerve fibers occurs in those structures the peripheral fibers of which are often stimulated at the same time, their function being thus simultaneously excited.

Cutaneous Innervation of the Head.—The skin of the head receives its sensory supply from several nerve roots, especially the fifth and the upper cervical nerves; in fishes from the first cervical nerve, and in man from the second cervical dorsal root, for in man the first cervical has no dorsal root.

As all these nerves in their peripheral distribution partly overlap each other, it is evident that they are often stimulated at the same time. Centrally, their fibers meet chiefly because of the structure of the trigeminus sensory root which, after its entrance into the oblongata, has a long course, descending to the sensory region of the first (or second)

* Submitted for publication, Oct. 24, 1929.

* In this survey I desire to acknowledge the assistance rendered by Dr. F. Robbins, New York, who has furnished the majority of the translations. Without her aid, this year's work would not have been possible.

1. Ariëns Kappers, C. U.: Acta psychiat. et neurol. **2**:118, 1927.

cervical nerve, so that the neurons of all these nerves have a common center of termination in the upper cervical cord.

The vagus also plays a part, although small, in the sensory innervation of the head, although its chief fibers do not innervate the skin, but go to the gullet and the intestines. Simultaneous stimulation of the vagus with other nerves of the head occurs only in those branches of the vagus, which in man pass to the ear and the external auditory canal.

The separation of the two parts of the vagus—gullet-intestine fibers and sensory skin fibers—is explained by its two central terminations, as the vagus when it enters the oblongata divides into two groups, the greater of which (largely gustatory fibers in the cod) terminates in a mediodorsal part of the oblongata, while the other group—that of skin fibers—joins the skin fibers of the trigeminus and descends with it. Consequently, here occurs a central connection of the cutaneous fibers of the head, evidently based on an associated function. This connection of fifth and vagus is also because of simultaneous stimulation.

Sense of Taste.—In man, the sense of taste is principally localized in the tongue and palate innervated by the seventh (facial) and the ninth (glossopharyngeal) nerves which enter the central nervous system at a distance from each other, but as their peripheral branches on the tongue partly overlap they are usually stimulated at the same time. Consequently their central axons join in the oblongata, all ending in one center, in which (in fishes) the taste fibers of the vagus also end. This is accomplished by the fibers of the seventh (facial) root descending in the medulla a considerable distance in order to reach the centers of the ninth and tenth nerves.

This process (neurobiotaxis) is seen in still another form in connection with taste.

It is well known that the organs of taste and smell are often stimulated simultaneously. Thus if one sips a fragrant cordial it is tasted and smelt simultaneously. For this purpose, an anatomic correlation is established between the centers of smell and taste, a connection that is the more striking as smell is innervated by the first nerve and taste by the seventh, ninth and tenth nerves and their centers lie at a great distance from each other in the brain.

However, the tertiary (central terminal) connection between these two centers occurs in fishes, in which taste and smell are more developed than in man. In them a bundle, which originates in a secondary taste center of the oblongata, passes through the midbrain and ends in a part of the hypothalamus (Herrick), where the tertiary olfactory tracts from the forebrain also end.

The taste sense center itself is stimulated by impulses from the water in which the animal lives and by the gills, which are saturated

with oxygen or with metabolic products of respiration. The motor impulses of the gills are transmitted by short neurons to the motor seventh nucleus; and as the motor nucleus occupies the place corresponding to the strongest reflex tracts, the motor center of the seventh lies very near the taste sensory nucleus at the dorsal side of the brain stem.

On the other hand, the rodent presents a different location of the motor seventh nucleus.

In the rodent the sensory fibers of the mouth, nose and eyes run in the trigeminus; a secondary center of the cochlea (causing reflexes of the ear muscles) lies at the base in the oliva superior, while the optic reflexes (for the closing of the eyes in strong light) are also situated at the base of the oblongata.

As a consequence, the chief part of the motor nucleus of the seventh of the rodent has a more central position near the cochlear ear muscle and optic motor light reflex tracts, and also near the decussation of the facial pyramid, which transmits cortical impulses to this nucleus. Only a very small part of the seventh nucleus has kept a dorsal position near the taste center.

Another motor nucleus which changes its position because of the chief reflex system which dominates its function is the abducens (sixth) nucleus.

The outward rotation of the eye depends largely on two forms of impulses: (*a*) from the organ of equilibrium, and (*b*) from the eye itself. Both sets of impulses take their course dorsally and ventrally in the oblongata.

In those fishes in which the dorsal tracts dominate, the abducens nucleus has a dorsal position in the medulla; in those in which the ventral reflex paths dominate, it has a ventral position.

Thus in the fish, the facial (seventh) nucleus (a motor center) acquires another position when the gill arches are changed. However, with an alteration of the sensory relations in the gill region only the motor nuclei of the gill arches and no motor cells of eye movements change their position.

In mammals and in man, the abducens nucleus also has a dorsal position, but it lies a little more laterally, under the influence of Deiters' nucleus.

Acoustic Innervation.—The acoustic organ in lower animals is very poorly developed, but the vestibular and lateral line senses are strongly developed. Most fishes are deaf, and the hearing which some possess is probably limited to the perception of noise.

Hearing really commences among vertebrates in *Amphibia* and increases in reptiles and birds, to reach its acme in mammals, particu-

larly in some rodents and terrestrial carnivora in which the acoustic apparatus acquires a high degree of development.

This gradual evolution of the peripheral acoustic organ has a striking influence on the situation of the acoustic region in the central nervous system.

In reptiles and birds, both central acoustic nuclei (the nucleus lateralis or angularis, and medialis or magnocellularis) lie at the dorsal side of the oblongata. The cochlear nerve approaches these nuclei from below.

In mammals, with the enlargement of the cochlea and of the cochlear nerve, the auditory nuclei commence to shift ventrally more and more, and finally acquire a position near the base of the medulla. This migration starts in the monotremes (among which are duck-moles) and reaches its maximum in rodents, bats, carnivora and primates.

Shifting of Vestibular Nuclei.—A similar process of shifting occurs in the nuclei of the cerebellum because of their association with the vestibular apparatus. The cerebellar nuclei, which originally are derived from, and are connected with, the vestibular nuclei, shift more and more into the cerebellum itself. This is because the vestibular nuclei of the oblongata receive impulses from the cortex of the cerebellum.

A part of these nuclei, which in lower vertebrates still lie in the dorsal edge of the oblongata, in higher vertebrates shift into the cerebellum forming the nucleus tecti (or fastigii) and the dentate nucleus. This process starts in reptiles; however, in birds the nucleus tecti and dentate nucleus are still contiguous with the vestibular nuclei of the oblongata. Only in the higher mammals are they entirely separated from nuclei, being completely embedded in the white matter of the cerebellum, as in mammals Deiters' nucleus and the nucleus radialis vestibularis descendens receive fibers from the cerebellar cortex (Hakenbandel).

With the increase of the cerebellar cortex and of the stimulations radiating from the Purkinje cells, the nuclei shift more and more in the direction of the cortex, so that the nucleus tecti (which is chiefly connected with the cortex of the anterior lobe) extends more frontally than the dentate nucleus which lies nearer the cerebellar hemispheres, sending (in rabbits) separate strands into the flocculus.

Primitive Bilateral Equilibrium Stimulation by Decussation.—An example of how bilateral simultaneous impulses cause a central union of bilateral projections necessitating partial decussation is the central connections of the organs of equilibrium (statocysts) in an invertebrate (*Pterotrachea,* a family of shell-less heteropods). In these the statoliths (stones) which stimulate the hair cells of the macula press on the right wall of both statocysts when the animal turns over to the right side; i. e., the stones stimulate the outer half of one statocyst, while they

stimulate the inner half of the other. This implies that the fibers of the outer half of one statocyst must centrally join the fibers of the inner half of the other statocyst, and this occurs. Consequently, the central connections become such that the nerves of both statocysts exhibit a semidecussation. This has the effect that both statocysts collaborate centrally in one ganglion at each change in the position of the animal and consequently the animal is enabled to correct its position more quickly.

"THE EMBRYOLOGY AND NEUROHISTOLOGY OF SPHENOPALATINE GANGLION CONNECTION" [2]

Cephalogenesis has led to a grouping of similar components together within the brain stem.

In mammals the fifth nerve has peripherally mostly sensory, and the seventh nerve mostly motor, components, although in lower vertebrates both nerves have mixed contents.

The sphenopalatine ganglion embryologically is formed by migration of cells from the fifth and seventh ganglion. It may retain its connections with the glossopharyngeal and the vagus.

The principal connection of the sphenopalatine ganglion with Meckel's ganglion is the vidian nerve made up of (*a*) the great superficial petrosal and (*b*) the great deep petrosal.

The great superficial petrosal is homologous with the palatine branch of the facial nerve of lower forms.

The great deep petrosal in man is made up of unmyelinated postganglionic sympathetic fibers from the superior cervical ganglia but it contains a considerable number of myelinated fibers from other sources, perhaps from the petrous ganglion of the glossopharyngeal, which probably reach the great deep petrosal by Jacobson's nerve and the small deep petrosal. Such connection would make the sensory fibers of the great deep petrosal homologous with the palatine branch of the glossopharyngeal of fishes. The sphenopalatine branches of the maxillary nerve going to the palatine nerves *pass through* the sphenopalatine ganglion without interruption.

Fenton and Larsell have also found a small branch corresponding to the ramus cutaneus facialis in a human fetus, thus confirming Rhinehart's observation of the sensory connection of the external auditory canal and tympanic membrane area with the vagus and facial nerves (in the mouse), as both the chorda tympani and the great superficial petrosal contain afferent fibers the cell bodies of which are located

2. Fenton, R. A., and Larsell, O.: Mechanism of Pain Transmission in Certain Types of Otalgia, Ann. Otol. Rhin. & Laryng. **37**:739 (Sept.) 1928.

in the geniculate ganglion and afferent fibers which *pass through* the ganglion without connection with its cells.

Carpenter expressed the belief that sympathetic tissue is distinguished by "typical multipolar cells encircled by fine fibrillary end nets." And Fenton and Larsell's investigations have confirmed the sympathetic character of the sphenopalatine ganglion, tracing back the sympathetic fibers to the great superficial petrosal nerve in which they constitute fibers of typical preganglionic character. They demonstrated that post-ganglionic fibers leave the sphenopalatine ganglion by the various nerves which radiate from it.

The geniculate ganglion belongs to the craniospinal series of sensory ganglions as its cells are of the unipolar sensory type. Fenton and Larsell found fibers of the sensory type in the great superficial petrosal.

They demonstrated that in the sphenopalatine ganglion there exists the usual condition for reference of pain from a visceral region to a body surface; viz., that ganglionic cells of afferent fibers of both areas are located in proximity in the same ganglion (geniculate) while centripetal processes of both sets of cells pass together into the bulb. Consequently, vasomotor changes, infiltration, infection or pressure in the sphenopalatine sensory distribution may stimulate the palatine seventh (facial) and other visceral sensory fibers; such impulses, passing through the great superficial petrosal to the geniculate ganglion come into relation with the somatic sensory cells and fibers of the ramus cutaneus facialis, and thus pain may be transformed outward to the auricular and mastoid regions.

Fay [3] sectioned intracranially the vagus (tenth) and the glossopharyngeal (ninth) nerves, and came to the conclusion that the glossopharyngeal in man is a special nerve (of taste and sensation), while the vagus (tenth) is a combined motor and sensory nerve—its sensory fibers going to the pharynx, larynx, external ear and over the mastoid region, the latter by its own direct branches and also indirectly through its extracranial portion by way of the ramus auricularis. Whatever sensory fibers pass in the extracranial portion of the glossopharyngeal nerve come from the vagus.

The vagus supplies sensation to the external ear, as well as to the pharynx and larynx. The vagus is responsible for the "gag" reflex.

Fay also expressed the belief that the reflex pain of so-called glossopharyngeal neuralgia, viz., pain in pharynx or in ear, is a manifestation of (ninth) vagus involvement in its sensory pharyngeal branch. A better term would be vagal-auricular-pharyngeal neuralgia.

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3. Fay, Temple: Observations and Results from Intracranial Section of the Glossopharyngeus and Vagus Nerves in Man, J. Neurol. & Psychopath. **8**:110 (Oct.) 1927.

Cortical Deafness.—Central deafness [4] is extremely rare because it necessitates bilateral foci. The central auditory tract passes from the periphery by way of the ventral acoustic nucleus situated in the lateral eminence of the rhomboid fossa of the medulla. From the nucleus, the auditory tract goes in two separate portions to the corpus geniculatum mediale. The medial portion passes through the corpus trapezoideum of the pons where it intersects with the tract of the opposite side; the lateral portion passes through the lateral fillet, divides into a bundle which goes to the corpus quadrigeminum inferius of the opposite side, and from here to the corpus geniculatum mediale where it meets the fibers which pass through the corpus trapezoideum. From the medial geniculate body, the auditory tract passes by projection fibers to the auditory cortex in the temporal lobe. Each acoustic nucleus is thus connected with both auditory cortices by several separate fiber strands.

Deafness can be produced by a lesion in four different regions, which may be clinically distinguished as follows: (1) lesions in the labyrinth and (2) lesions in the postlabyrinthine neuron; the latter cause gradual deafness by atrophy of the cochlear nerve. Postlabyrinthine deafness is generally unassociated with other phenomena; when it occurs in tabes there are other tabetic symptoms. (3) Foci in the pons, peduncles or corpora quadrigemina lead to bilateral hardness of hearing or total deafness. This deafness occurs suddenly when due to hemorrhage, or slowly when due to tumor. Localization may be possible when there are accompanying symptoms of the cranial nerve nuclei involvement. At this level all the fibers of the auditory tract are usually not involved; consequently there usually exists hardness of hearing, not total deafness. (4) Subcortical or cortical foci in a unilateral lesion of one temporal lobe result in impaired hearing, which is often only transitory. Only bilateral foci give total deafness. The associated symptoms of sensory aphasia and hemiplegia are characteristic.

The discovery of the location of the auditory cortical sphere is due to Flechsig, who showed that the auditory radiations (derived from the corpus geniculatum) pass directly under the insular cortex and terminate in the anterior transverse convolution of the temporal lobe, the cortical area being restricted to the anterior transverse convolution and the adjacent 1 to 2 cm. of the first temporal convolution.

In all cases of central deafness reported in the literature, which have come to autopsy, a bilateral destruction of the transverse convolution or of the adjacent medullary layer could be demonstrated; but the lesions were so extensive that portions of the temporal and parietal lobes were also involved. In none of the cases was the lesion limited

4. Misch, W.: Cortical Deafness, Ztschr. f. d. ges. Neurol. u. Psychiat. **115**:567, 1928.

to Flechsig's auditory sphere, viz., in the transverse convolution of the temporal lobe, so that clinical confirmation of Flechsig's investigation has not been furnished prior to Misch's case which is characterized by the lesion being circumscribed to this area at least on one side. It affected the median portion of the anterior transverse convolution.

The pathologic course was as follows: During septic endocarditis (viridans sepsis), an embolism lodged in the left temporal lobe, which subsequently led to a slowly progressive hemorrhage into the white substance of the entire left hemisphere. There were sensory aphasia and a gradually advancing right-sided hemiplegia. No auditory disturbances were demonstrable. About ten days later, a small hemorrhage occurred in the depth of the sylvian fissure on the right (the opposite) side, due to septic embolism of the sylvian artery. This resulted in the destruction of the cortex of the median portion of the anterior transverse convolution and the adjacent white substance, to a depth of a few millimeters. As the left temporal lobe with the left auditory tract was already destroyed, the result was total bilateral deafness, which persisted until the patient's death a few days later. The fatal termination was due to rupture of the left-sided hemorrhage into the ventricles.

Although the destruction of the left hemisphere was extensive and involved the white substance of the entire temporal lobe, the right-sided lesion was localized to the cortex. It was the size of a cherry pit and was situated in the posterior end of the sylvian fissure directly behind the island of Reil. The septic embolism within the posterior portion of the artery of the fissure of Sylvius evidently has not completely occluded the artery, as the posterior area supplied by the artery remained intact. Thus only the most median portion of the anterior transverse convolution was destroyed. It could not be shown that the cortical area actually corresponded to the full extent of Flechsig's auditory sphere. But the lesion in the median portion of the anterior transverse convolution involved a sufficient depth to have interrupted the auditory radiation passing under it.

Misch's summary: Central deafness is due to a bilateral destruction of the cortical auditory sphere or its medullary radiation. The clinical course is characterized by two more or less separated attacks, which correspond to the foci on the two sides, associated with unilateral or bilateral hemiplegia and usually accompanied by sensory aphasia. The second attack leads to sudden deafness, which is always complete and bilateral. This case of cortical deafness is unique in that on one side a very small hemorrhage due to septic embolism of the hindmost portion of the sylvian artery almost exclusively destroyed the cortex of the median portion of the anterior temporal transverse convolution with the subjacent marrow.

Auditory Hallucination in Cerebral Tumors.—Hallucinations of smell, taste and vision are of frequent occurrence in cases of tumor of the brain, but Cushing has remarked that auditory disturbances are conspicuous by their absence.

Courville [5] examined the records of 412 verified cases of tumor of the brain in Cushing's clinic.

5. Courville, Cyril B.: Auditory Hallucinations Provoked by Intracranial Tumors, Arch. Neurol. & Psychiat. **19**:558 (March) 1928.

The cases showing auditory hallucinations may be divided into three groups: (1) tumors of the frontal lobe, (2) tumors of the temporal lobe and (3) multiple tumors of the brain. Among the frontal lobe tumors were six cases in which the patients had definite auditory hallucinations. In all but one, the hallucinations were those of voices, associated with evidence of great increase of intracranial pressure and definite mental changes amounting to a psychosis. There were four cases of temporal lobe tumors with definite auditory phenomena. In one case of suspected tumor of the temporal lobe auditory hallucinations developed after a subtemporal decompression and probing of the lobe in an attempt to locate the tumor; the phenomena probably resulted from trauma. Two cases of multiple metastatic tumors of the brain presented auditory phenomena.

FERMENT IN CEREBROSPINAL FLUID

Intoxication from Absorption of Dead Cerebral Tissue by the Cerebrospinal Fluid Into the Blood.[6]—1. Experimental freezing of small areas of the dog's cerebral cortex causes epileptic convulsions after from two to five hours, followed by death in from twelve to fifty hours. 2. With the exception of the motor region, the result is the same regardless of the portion of the cortex frozen. 3. If the frozen part is removed immediately after freezing, no symptoms result. 4. If, on the other hand, the frozen part is not excised until after the onset of symptoms, death may result. 5. Transplantation of a part of the frozen brain to the subdural space of a healthy dog induces symptoms of epilepsy and causes death. 6. Subcortical symptoms are the first to appear after freezing and the last to disappear.

Speransky expressed the belief that an autoneurotoxin is formed, which passes into the blood, producing hyperkinetic symptoms, because an intense motor excitement is produced by the injection of from 150 to 300 cc. of defibrinated blood from the ill animal into the blood of a healthy dog. These products reach the cerebrospinal fluid in large quantities. Withdrawal of the fluid immediately before the freezing of the brain causes a delay in the reaction. If the withdrawal is done two or three hours after the freezing, the development of the symptoms is not prevented, and the animals die.

Influence of the Cerebrospinal Fluid on Physiologic and Pathologic Processes in the Brain.—On the supposition that the dissolution of devitalized cerebral tissue took place through the agency of the cerebrospinal fluid, Speransky performed the following experiments:

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6. Gantt, W. Horsley: Recent Work of Pawlow and His Pupils; Conditioned Reflexes; Sympathetic Nervous System (Orbeli); Epilepsy and Cerebrospinal Fluid (Speransky), Arch. Neurol. & Psychiat. **17**:514 (April) 1927.

Pieces of the brain of the dog were placed in a fresh extract of cerebrospinal fluid of the dog in a thermostat. After twelve hours the pieces of the brain in the cerebrospinal fluid disappeared, the fluid apparently dissolving them. After from twenty-four to thirty-six hours, the turbid fluid contained only a sediment. Similar pieces of brain cortex placed in blood serum and physiologic solution of sodium chloride began to dissolve only at the end of the third day, and even after five days the dissolution was not complete. White of egg, blood fibrin and boiled brain are not changed in the cerebrospinal fluid. Only the fresh or frozen brain is dissolved, and of the dissolving pieces of brain, only the brain substance itself is split up; the pia and blood vessels maintain their resistance for a long time after the brain substance has disappeared entirely.

Speransky deduced from his experiments that only those albumins which enter the composition of the brain tissue are decomposed quickly by the cerebrospinal fluid. The other albumins are not changed or are changed slowly. As the bacterial toxins appear to be close in their chemical nature to the noncerebral tissue albumins, they are not quickly destroyed in cerebrospinal fluid.

Speransky suggested that the autodigestion of dead brain by the cerebrospinal fluid is the same as the autodigestion of dead mucous membrane of the stomach by the gastric juice.

He concluded: If destruction of the brain substance occurs in disease, the products of this destruction are transported into the cerebrospinal fluid, and through it poison the brain. To this is due the development of diffuse encephalitis.

The undamaged pia mater gives better protection to the brain substance than the ependyma; thus subcortical often occurs earlier than cortical destruction.

II. ADVANCES IN THE KNOWLEDGE OF THE RELATIONSHIP BETWEEN SPHENOID SUPPURATION AND CEREBRAL DISEASES

SUBMUCOUS PHARYNGEAL HYPOPHYSIS AND THE ANTERIOR LOBE OF THE PITUITARY

Their Relation to Adenoids and Diseases of the Nasopharynx.—Citelli [7] claims priority for having been the first to describe the existence of an endomucous portion of the pharyngeal hypophysis, to which he gave the name of vertical or endomucous pharyngeal hypophysis. He was also the first to point out the existence of the close circulatory relations between the pharyngeal hypophysis and the mucosa of the pharyngeal roof. He found that persistence of the craniopharyngeal canal, especially in children and adolescents, is not so rare as believed,

7. Citelli, S.: New Experimental and Clinical Data Showing the Influence of Diseases of the Nasopharynx and the Sphenoidal Sinuses on the Hypophysis, Arch. internat. de laryng. **7**:129, 1928.

as in twenty cadavers the canal was completely open in three and partially closed in one. Anatomic changes of the cerebral hypophysis have been observed as a sequel of diseases of the pharyngeal vault. Citelli said there can be no doubt as to the action of certain diseases of the rhinopharynx and the sphenoidal sinuses on the hypophyseal system. He described a psychic syndrome which consists of somnolence (sometimes insomnia), defective memory, mental sluggishness, distaste for work and difficulty of concentration. This syndrome is encountered in about from 35 to 40 per cent of patients with adenoids. Youthful persons with adenoids and intellectual deficiency sometimes present signs of hypophyseal effeminacy, especially hypotrichosis, absence of normal development of hair, and a feminine arrangement of the pubic hair being relatively often met with in youthful subjects with adenoids. Besides developmental disturbances, asthenia is not uncommonly encountered in adenoid vegetations and diseases of the pharyngeal roof. This asthenia disappears under treatment, and Citelli expressed the belief that it must be of hypophyseal character. He claimed excellent results from the use of whole hypophyseal extract or extract of the anterior lobe alone.

Citelli's [7] later experiments were on rabbits. He caused slight but frequently repeated irritation of the pharyngeal vault for months and even for years. The irritations were applied on the soft portion of the palate, into which in rabbits opens the inferior extremity of the craniopharyngeal canal. The conditions were thus fairly similar to human pathology during the first fifteen years of life, in which the craniopharyngeal canal persists in from 10 to 15 per cent of the cases. Four young rabbits were subjected to treatment; one, 8 months of age, received applications of tincture of iodine. On the three other rabbits, rather superficial burns were produced with silver nitrate. The first rabbit remained under observation for twenty months. The tincture of iodine never produced macroscopic lacerations of the velum, but after killing the animal, the cerebral hypophysis showed an increase of the eosinophils. The three rabbits subjected to superficial siver nitrate burns on the velum were also without macroscopic inflammatory changes of the nasopharynx, but had distinct histologic changes in the central hypophysis. These changes were in proportion to the severity of the inflammatory stimulus, the duration of the treatment and also apparently to the age at which the irritation was begun.

According to Citelli, these experimental data are of physiopathologic importance, because irritation by sphenoidal disease or adenoids in an infant with a previous craniopharyngeal canal must influence not only the pharyngeal hypophysis but also the central hypophysis and possibly also the infundibulum and the neighboring parts.

Histologic Studies of the Influence of Adenoids on the Hypophysis.—Slobodnik [8] carried out histologic investigations as to the connection between the pharyngeal tonsil and the pharyngeal hypophysis, to

8. Slobodnik, T.: Pharyngeal Hypophysis and Pharyngeal Tonsil, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:165, 1928.

determine the influence of adenoidism on the latter; as he thought that because the pharyngeal tonsil is situated in the vicinity of the pharyngeal hypophysis, the resulting improvement after an adenectomy might be due to the simultaneous removal of the latter. Histologic examinations were made on sixty-five specimens obtained by adenotomy. No trace of pharyngeal hypophysis tissue was found in any specimen.

EMBRYOLOGY OF THE PHARYNGEAL HYPOPHYSIS

Its Relation to the Pharyngeal Tonsil and Intracranial Hypophysis.—In man are found: (1) the cerebral hypophysis or pituitary, (2) the pharyngeal hypophysis and occasionally some (3) hypophyseal "germs."

The nasopharynx in the embryo and new-born infant contains the common "anlage" of the pharyngeal tonsil and pharyngeal hypophysis; they later become differentiated. Embryogenetically, the pharyngeal hypophysis is closely related to the cerebral hypophysis and contains similar structures. The anterior portion of the cerebral hypophysis develops from the outer hypophyseal pouch (Rathke's pouch) and passes from the nasopharynx through the craniopharyngeal canal in the sphenoid bone into the cranial cavity. In its further development, it forms the anterior lobe of the pituitary. The posterior lobe (neurohypophysis) develops from the base of the mesencephalon. As these portions originate from different embryonic anlage, they also differ histologically. The anterior lobe consists of chromophobe cells (main cells) and chromophil cells (acidophilic, eosinophilic, basophilic cells), and in addition contains remnants of pavement epithelium. In the course of development, the craniopharyngeal canal disappears; traces of it may remain in the sella turcica or in the nasopharynx, or there may be hypophysis cells, differentiated or undifferentiated. Some writers think these remnants, hypophyseal "germs" or "nests," form the material for tumors.

In addition, in man there is constantly found, in one part, hypophysis-like tissue of the nasopharynx. This pharyngeal hypophysis-like tissue is closely related histologically and embryonically with the anterior lobe of the hypophysis. It is named the pharyngeal pituitary or hypophysis because of its anatomic situation and its histologic structure. This constant structure has but recently been discovered. Its function is not known.

In the new-born child, the anterior lobe of the cerebral hypophysis has typically differentiated cells. The cells of the pharyngeal hypophysis are differentiated later, as in the embryo and in the new-born infant they form a compact undifferentiated mass; its alveolar structure and complete cell differentiation only become recognizable during the first

year of infant life. The pharyngeal hypophysis is situated in the median line at the upper posterior border of the vomer where the latter articulates with the sphenoidal bone. It has the form of a strand, sometimes with thickenings at one or both ends. Its size varies from 2 to 4 mm. in embryos and children to 6, 7 or 9 mm. in adults; its thickness is from 0.5 to 1 mm. and it has a direction upward and backward, passing between the mucosa and the periosteum. Posteriorly, it may extend beyond the adenoid tissue. It has no capsule. It is microscopically visible in sections when stained. It is separated from the surrounding tissue by connective tissue, but microscopically it differs from the surrounding tissues. Its upper end often comes in close contact with the adenoid tissue of the nasopharynx. Its stroma is delicate and sometimes slightly thickened in aged persons. Like the cerebral hypophysis, it has an alveolar structure; it has chromophobe cells, main cells, and chromophil cells, eosinophilic and basophilic cells. Thus the pharyngeal hypophysis and cerebral hypophysis are qualitatively similar, although the proportion between the eosinophils and the basophils is different.

In all cases, there is a close connection between the pharyngeal hypophysis and the pharyngeal tonsil. Specimens from embryos as well as from adults show that the pharyngeal hypophysis may lie above the adenoid tissue. Sometimes the pharyngeal hypophysis extends to the anterior border of the pharyngeal tonsil and often comes in close contact with it. There also exist relations between the vascular systems of both organs.

As the result of not finding any hypophysis-like tissue in the sixty-five cases of adenoids, Slobodnik did not think that the mental improvement that follows adenectomy is due to removal of the pharyngeal hypophysis, but that it may be due to the relief of venous stasis in the cerebral pituitary.

MENTAL SYMPTOMS AND SPHENOIDAL SUPPURATION

Adam[9] said that it cannot yet be admitted that there are cases of toxic psychosis resulting from sinusitis; for a positive conclusion a large number of cases of association of sinusitis with mental symptoms need to be recorded, and the sequence of cure of insanity following operation for sinusitis must also be shown. In each of his two cases the sinusitis preceded the mental symptoms. The first patient had a right-sided pansinusitis; all the cavities were drained, but the patient died some years later in an asylum. The second patient had a bilateral pansinusitis; all the cavities were dealt with and are practically well, but the patient is still in an asylum.

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9. Adam, James: Chronic Nasal Sinusitis with Mental Symptoms, Brit. M. J. **2**:794 (Nov.) 1928.

Adam very opportunely suggested that it is greatly to be desired that the mental symptoms associated with chronic sinusitis and those associated with frontal lobe abscess be carefully observed with a view to clinical differentiation. He asked the pertinent question, "What are the mental symptoms of 'toxic psychosis' as distinguished from those of frontal lobe abscess?"

On the other hand, Pickworth [9a] reported a case of "Perforation of the Pituitary Fossa by a Septic Lesion of the Sphenoidal Sinus Associated with Recent Epilepsy and Insanity," and stated that infective lesions of the sphenoid sinus are often found both clinically and after death in acute and chronic cases of mental disorder. Some postmortem specimens show a discoloration of the pituitary gland or the bony partition between it and the sphenoidal sinus. Sinus disease is also found in cases of epidemic encephalitis. It is therefore not improbable that in the course of sphenoidal sinusitis micro-organisms or their toxic products penetrate the bony wall and produce disturbances of pituitary function of neighboring nervous structures and cerebrospinal fluid, which may in turn be responsible for the symptoms of the mental disorder and epilepsy.

Pickworth said that the cerebral structures adjacent to the pituitary are of importance in certain of the higher metabolic functions (Cloake), which include normal temperature regulation, sex gland activity, sugar metabolism, and obesity, polyuria, etc. Adie expressed the belief "that the pituitary body and the nucleus hypophysis (nucleus supra-opticus) and adjacent structures in the 'tween-brain work together as an endocrine nervous system," and further that idiopathic narcolepsy is a disorder of this system. This is contrasted with its clinical opposite, exophthalmic goiter, in which the 'tween-brain disorder is considered to be of more importance than that of the thyroid gland. In support of this view are the observations of Bailey and Bremer that apathy and drowsiness have been frequently recorded after experimental puncture of the tuber cinereum, and that hyperthermia is a common sequel to any experimental lesion of the tuber. Pette expressed the belief that there is a sleep center in the gray matter of the floor of the third ventricle.

Pickworth's patient had mental symptoms: hallucinations, paranoid suspicions, disorientation, noisy, resistive, impulsive behavior and defective memory.

The postmortem examinations showed the pituitary gland surrounded by a thickened capsule which was not adherent to the floor of the fossa, but was separated from it by a quantity of mucinous fluid, which could be seen slowly draining through a hole communicating with the right sphenoidal sinus; the latter also contained a quantity of similar fluid. The hole was judged to be of long standing, on account of the bony changes in the neighborhood.

9a. Pickworth, F. N.: J. Laryng. & Otol. **43**:186 (March) 1928.

There were areas of small but not very recent hemorrhages in the capsule of the gland, and altered blood pigment indicated older hemorrhages. Some colloid material was found in the capsule. Near the colloid masses and in other parts of the capsule were bodies resembling organisms which apparently filled the minute lymphatic vessels or cerebrospinal fluid spaces. These bodies were gram-negative. The same organisms were found also as high up as the pituitary stalks, but not in the brain substance itself.

Pickworth expressed the belief that it is probable that they were originally virulent cocci which became biologically inhibited by the tissue in which they were growing. Such biologic inhibition of bacterial growth by the patient's own immune serum has been shown experimentally.

A section of the optic nerve at the level of the anterior clinoid process showed gram-positive micrococci in the dural sheath, indicating considerable extension of the septic process subdurally.

I can attest to the contention of Pickworth as to the frequency of bony discoloration in the roof of the sphenoid sinus, as on several occasions I have noted a peculiar discoloration of this region in patients dying of intracranial suppuration, without macroscopic evidence of sphenoidal sinus disease. It is possible that this discoloration is the first stage of necrosis as in Ivanovski's case.

Ivanovski [10] reported as an autopsy observation "neurosis of the hypophysis as a complication of otogenic sinus thrombosis" in a patient who died of septicemia resulting from thrombosis of the left sigmoid sinus, jugular bulb, superior and inferior petrosal sinus and bilateral thrombosis of the cavernous sinus. The bony necrosis is referred to the thrombosis of the sinus surrounding the hypophysis, thus rendering venous drainage impossible. The author called attention to the unique character of this case, which appears to be the first reported in the literature.

OPTIC NEURITIS FOLLOWING SPHENOID SINUSITIS

Optic neuritis following sphenoid sinusitis is gradually passing out of the domain of speculation and snapshot surgery into that of definite clinical medicine.

There are undoubtedly two separate lesions which, in the presence of the proper anatomic relationship of the ethmosphenoidal mucous membrane to the optic nerve sheath, will give impairment of vision, viz., actual infiltration of the nerve as now shown by Pickworth, and neuritis from toxemia.

Daily repeated examinations of the visual field will in the future aid in the differentiation.

I have summarized my own experience in an article on "Localizing Value of Ophthalmic Examinations in Suppurative Diseases of the

10. Ivanovski, S.: Russk. Otol. **21**:269, 1928.

Brain,"[11] in which I stated that suppuration in the bone surrounding the optic canal from spheno-ethmoid disease causes a neuritis which is evidenced (*a*) by swelling of the nerve head; (*b*) a central scotoma accompanied by rapidly failing vision, associated with (*c*) pain on pressing the eyeball back into the orbit and on movement of the eyes.

I suggested that the neuritis, with its accompanying central scotoma and rapidly failing vision, is probably due to the anatomic adhesion of the nerve's sheath to the optic foramen; an inflammation of the bony wall resulting in a "biting" of the nerve and an involvement of the macula bundle. The pain on pressure and during ocular movements is due to the presence of an infective process in the bone at the margin of the optic foramen thus involving the annular ligament of Zinn, to which is attached the external ocular muscles, the ligament itself being attached to the bony wall.

In the combined experience of the staff of the Newark Eye and Ear Infirmary, these four factors, (*a*) cold in the head, (*b*) swelling of the nerve head, (*c*) central scotoma and (*d*) retro-ocular pain on pressure and during ocular movements, were present in all of the three cases in which the immediate result of opening the spheno-ethmoid sinuses was sufficient to warrant the belief that the nasal sinus suppuration was the cause of the failure of vision.

Doesschate[12] expressed the belief that a disease of the sphenoid sinus may exert a toxic influence on the optic nerve and induce an enlargement of the blind spot.

1. A patient had attacks of dyspepsia and swelling of the right half of the face, with a high fever. Nasal examination showed acute rhinitis and an ulcerated septum. However, he had an enlargement of the blind spot. Postmortem examination revealed edema, infiltration, thrombotic veins in the mucosa of the sphenoid cavity; osteomyelitis of the adjacent bone, and thrombotic veins in the vicinity of the cavernous sinus. There was slight edema of the optic nerve sheath.

2. A patient had chronic perisinusitis. There was no anamnesis or clinical data for this case. The patient died of pneumonia. Postmortem examination revealed purulent infiltration in the mucosa of the sphenoid sinus, and diplococci in the marrow of the septum and in the dural sheath of the optic nerve.

On the other hand, Watson-Williams'[13] case presented no involvement of the visual field or enlargement of the blind spot. Three weeks after a sphenoid sinus operation, the vision had improved, associated with a very definite lessening of the optic neuritis.

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11. Eagleton, W. P.: Localizing Value of Ophthalmic Examinations in Suppurative Diseases of the Brain, J. A. M. A. **92**:713 (March 2) 1929.

12. Ten Doesschate, G.: Pathologico-Anatomical Findings in Rhinogenic Optic Nerve Affections, Klin. Monatsbl. f. Augenh. **80**:831, 1928.

13. Watson-Williams, Patrick: Optic Neuritis Following Sphenoidal Sinusitis Located by the Differential Exploratory Test, Brit. M. J. **2**:1030 (Dec. 8) 1928.

INTRACRANIAL COMPLICATIONS FROM LATENT SPHENOID SUPPURATION WITH OUTSPOKEN OTITIS

The frequency of a fronto-ethmo-sphenoidal suppuration (which is latent as to outspoken symptoms) being associated with an otitis, which is the apparent cause of an intracranial complication, is too frequently disregarded by the otologist. On numerous occasions (once at least during the past year) I have been humiliated to find at post mortem that a collection of pus filled the sphenoid when all my surgical efforts had been directed to the neighborhood of the ear.

Bombelli [14] reported "A Case of Cerebral Abscess Derived from Latent Sphenoidal Sinusitis in a Patient with Grave Otitic Complications" which is noteworthy on account of the insidious appearance of the grave inflammatory process in the sphenoidal sinus, which was entirely concealed by the otitis. At the time of operation, there was an increased tension of the dura, but puncture was negative. The autopsy showed not an encapsulated otitic brain abscess as had been diagnosed, but a purulent encephalitis. The abducens paralysis was not referable to the otitis on the same side, but to a metastatic focus on the opposite side. Hypoglossus and vagus paralysis came from suppuration in the cervical region which extended to the base of the brain.

CAVERNOUS SINUS THROMBOPHLEBITIS

I was naturally pleased to find my work [15] understood in Europe where Christophe [16] has adopted and successfully applied my technic to a case of cavernous sinus thrombophlebitis.

An ambulatory patient had radiating pain which was interpreted as dental neuralgia. An abscess appeared under the first right premolar tooth. The pain in the right side of the face, behind the orbit and in the temporal region increased in severity. Floating black spots appeared suddenly in the right field; vision failed and by the third day the patient was totally blind in the right eye. She was now in bed with high temperature and chills. Exophthalmos slowly developed, with swelling of the upper lid, chemosis, complete immobility of the eyeball and blood in the anterior chamber. A diagnosis was made of thrombophlebitis of the cavernous sinus. Christophe said, "on consulting the book of Eagleton, a point of capital importance is emphasized, namely, that the sinus must be put completely at rest, just as a limb with phlebitis must be immobilized. For this it is necessary to stop the continuous pulsation of the internal carotid, which is engulfed in the sinus." Consequently, with the patient under local anesthesia, Christophe exposed the carotid artery and occluded it by a small Carrel clamp. The ascending process of the malar bone and the zygoma was exposed, and the external wall of the orbit

14. Bombelli, U.: Riv. oto-neuro-oft. **5**:142, 1928.

15. Eagleton, Wells P.: Cavernous Sinus Thrombophlebitis, New York, The Macmillan Company, 1926.

16. Christophe, L.: Thrombophlebitis of the Cavernous Sinus; Operation; Recovery, J. de chir. et ann. Soc. belge de chir. **27**:312 and 318 (Dec.) 1928.

was evulsed; the eye was enucleated and the sphenoidal fissure in the orbit was exposed. The wall of the sinus was opened and the thrombus seen but not removed, for as I have stated, the clot is a defensive process and must be carefully preserved. A drain was placed in contact with the clot in the cavernous sinus. The temperature promptly dropped to normal. On the sixth day, the clamp was replaced by a silk ligature, and the common carotid artery divided.

Christophe's case is the first one of thrombophlebitis of the cavernous sinus published in the French literature in which operation had been performed with recovery. Christophe thinks that the operative success is due the idea that I had expressed, viz., placing the sinus at rest through carotid ligature. "It provided a cure in the case of a patient who was apparently doomed."

Extended experience since writing the book on "Cavernous Sinus Thrombophlebitis," [15] has demonstrated that the cases favorable to this procedure are the subacute or chronic cases such as Christophe's. In the acute cases, the associated phlebitis of the inferior cerebral veins causes a meningitis as soon as the infective process enters the cranium because the cerebral veins run free without fibrous sheaths in the fluid of the large lake of the chiasmal cisterna; consequently infection is rapidly disseminated through the basal cisterna and over the cortex by means of the vascular and respiratory impulses. This was explained by me in a paper on "Cerebrospinal Fluid in the Diagnosis and Treatment of Infective Lesions," which I presented at the British Medical Society in Manchester on July 24, 1929.

VIRULENCE OF MENINGITIS THE RESULT OF THROMBOSIS OF THE CAVERNOUS SINUS

Guerra [17] reported the case of a patient who died of septic thrombophlebitis of the cavernous sinus following a purulent sphenoidal sinusitis with necrosis of the sphenoid bone. There was tenderness on pressure about the tragus as well as in the mastoid and sternocleidomastoid region, a fact which the author has not found mentioned in the literature. The disease ran an insidious course until a few hours before the patient's death, although extensive meningitis was found at autopsy. The latter also revealed purulent sinusitis of the posterior cells of the sphenoid, purulent meningitis, thrombosis of the cavernous sinus and inflammatory necrosis of the retrobulbar tissue. Histologic examination showed that in addition to infiltration of the meninges by exudate of fibrin and polymorphonuclear leukocytes, that some of the leptomeningeal vessels were thrombotic. The cerebral cortex was covered by a purulent exudate which in a general way followed the blood capil-

17. Guerra, P.: A Case of Thrombophlebitis of the Cavernous Sinus Due to Sphenoidal Sinusitis, Arch. di ottal. **35**:337, 1928.

laries. There were numerous hemorrhagic areas in the medullary substance. The pyramidal cells presented a necrotic appearance, with a poorly stained nucleus.

I wish to call attention to the presence of thrombosis of the veins and multiple hemorrhages of the cortex. It was doubtless by thrombosis that the meningitis originated.

Septic thrombosis of a cerebral vein was probably the pathologic cause in Luque's case [18] in which a fatal sinus thrombosis with meningitis and septicemia developed within a few hours afer the incision of a nasal furuncle; the patient was diabetic. Luque cautioned against operative intervention in purulent processes of the face involving the region of the ophthalmic and the superior pterygoid vein, as well as the veins of the foramen ovale, viz., those which drain the lips, eyelids, nose, cheeks, mouth and pharynx.

III. EAR AND NOSE IN THE DIAGNOSIS OF GENERAL DISEASE OF THE CENTRAL NERVOUS SYSTEM

CEREBROSPINAL RHINORRHEA WITH PNEUMOCEPHALUS SECONDARY TO FRACTURE OF THE SKULL TREATED BY THE UPRIGHT POSITION [19]

The patient received a fracture of the skull, about seven weeks before admission. He was comatose for several days, and was left with headaches and mental dulness, and a constant flow of clear fluid from the right nostril. When admitted to the hospital, he presented left-sided weakness, including a left facial paralysis of the central type. A roentgenogram gave no evidence of fracture, but revealed a large collection of air in the right lateral and also in the third and fourth ventricles. The patient was treated unsuccessfully by roentgenotherapy. An intranasal plastic operation was thought to be contraindicated because of the danger of infection. Strauss proposed that the head be kept in a position of absolute rest to allow the torn dura opportunity for proliferation, and thus to repair the rent through which the fluid escaped. This was done for a period of three weeks. There was complete cessation of the flow of the cerebrospinal fluid, with disappearance of mental symptoms, headaches and motor weakness. Three months later, the patient was well. Roentgenograms showed absence of the pneumocephalus.

CEREBRROSPINAL RHINORRHEA TREATED BY OPERATION WITH TINCTURE OF IODINE PACKS APPLIED EXTRADURALLY [20]

The patient had a long history of chronic infection in the accessory nasal sinuses, with acute exacerbations. About July, 1928, he noticed the escape of clear

18. Luque, E.: Contribution to the Pathogenesis of Thrombophlebitis of the Cavernous Sinus, Arch. de ocul. Hisp. **28**:850, 1928; Klin. Monatsbl. f. Augenh. **80**:850, 1928.

19. Bromberg, Walter: Cerebrospinal Rhinorrhea with Pneumocephalus Secondary to Fracture of the Skull, Arch. Neurol. & Psychiat. **21**:235 (Jan.) 1929.

20. Learmonth, J. R.: Cerebrospinal Rhinorrhea Treated by Operation with Extradural Tincture of Iodine Packs, Bulletin of Mayo Clinic, April 10, 1929, p. 115.

fluid from the right nostril; in October, 1928, there was a sudden discharge following an effort to lift a heavy weight. From October there was some discharge from time to time until the end of January, 1929, when cerebrospinal fluid leaked continuously from the nose for five weeks. The fluid was seen to be coming from the superior meatus and therefore was reaching the nose by way of either the ethmoidal air cells or the sphenoidal sinus, the most likely route being through the cribriform plate of the ethmoid bone. Learmonth turned back a flap above the forehead, avoiding the right frontal sinus, which had been operated on. He then elevated the right frontal lobe in its covering of dura, turning it backward and mesially until the region of the cribriform plate came into view. This was found to be occupied by vascular granulation tissue. Between this tissue and the dura, he inserted four packs soaked in tincture of iodine. The packs were removed one by one; the last was taken out nine days after operation. There was no drainage until the day of the report when there was a little return.

DEAFNESS IN CENTRAL NERVOUS DISEASES (SCHILDER'S DISEASE)

Globus and Strauss [21] reported four cases of progressive degenerative subcortical encephalopathy in infants and young children, all of whom had the typical decerebrate rigidity. Two of the patients had acute otitis media. Fairly typical features of Schilder's disease are an abrupt onset, a rather rapidly progressive course, characterized by mental deterioration, rapidly advancing spastic paralysis, epileptiform attacks, ocular palsies and other ocular manifestations, optic atrophy and blindness without optic atrophy followed by a rapid decline with fatal issue (within two years). Advancing blindness with or without optic atrophy, deafness and aphasia may develop at any stage of the illness, though most frequently early in the clinical course.

Stewart, Greenfield and Blandy,[22] in reporting their cases, said "the first symptom may be deafness of central origin which may precede other symptoms, such as blindness, by some months. They emphasized that in the differential diagnosis between Schilder's encephalitis and disseminate sclerosis, the chief clinical criteria are (1) the nature of the loss of hearing and (2) the progressive loss of vision.

Complete deafness is never met with in disseminate sclerosis, but unilateral impairment of hearing is not unknown.

EAR TESTS IN CENTRAL DEGENERATIVE LESIONS

In reading the literature on nervous diseases, I was struck with the absence of vestibular tests even in the presence of such suggestive symptoms as decerebrate rigidity (Schilder's disease) or dizziness. I

21. Globus, Joseph H., and Strauss, Israel: Progressive Degenerative Subcortical Encephalopathy, Arch. Neurol. & Psychiat. **20**:1190 (Dec.) 1928.

22. Stewart, T. G.; Greenfield, J. Godwin, and Blandy, Marjorie A.: Encephalitis Periaxialis Diffusa: Report on Three Cases with Pathological Examinations, Brain **50**:1, 1927.

am persuaded that the neurologist and the neurosurgeon are neglecting a very important cerebral mechanism. This, I believe, is largely due to the disappointment that followed the extravagant claims for the tests as localizing agents, made by otologists at the time when there was but an imperfect understanding of the vestibular nervous mechanism.

The following principles are now generally accepted: (*a*) The otolithic system is one of the most primitive of life's mechanisms, to which in vertebrates there is added (*b*) a very complicated semicircular canal system, which is attached to the central vestibular ganglia. From the regions of the vestibular nuclei there later develops the cerebellum. Much later in life's evolution the (*c*) auditory apparatus appears. There are thus three mechanisms connected with the human ear: (1) a primitive tropism for fundamental orientation—otolithic—with cerebral connections about which little is known, and for which no tests are as yet available, (2) a mechanism for orientation of the head with the muscles of the eyes and back—semicircular canal system—whose central anatomic pathways are in doubt but for whose integrity as a whole, there are available tests, and (3) a highly specialized nervous mechanism for hearing, the cerebral course of whose fibers are well known, but an exact localization of a lesion of which is frequently impossible—partly because of the association of the contralateral tracts, but chiefly because the understanding of sound is as yet vague.

I recently saw a surgeon fail to avail himself of the vestibular tests in surgical diagnosis at a large clinic; a patient with disseminate sclerosis was operated on for suspected tumor of the cord—a mistake which might have been avoided had the vestibular tests been made.

I believe that an increase in the duration of the induced nystagmus from turning is a constant early manifestation of disseminate sclerosis, and consequently is of the greatest diagnostic value.

As far as I am aware, few patients with cord tumor have been subjected to the turning or the caloric tests

SYPHILIS OF THE NERVOUS SYSTEM

Hassin's [23] contribution to the pathology and pathogenesis of tabes dorsalis is of interest to the otologist, although it does not mention deafness and involvement of the vestibular apparatus. His studies demonstrate that tabes is due to a proliferation of arachnoid cells into the subarachnoid spaces of the posterior roots of the spinal cord, thereby obliterating the arachnoid prolongations surrounding the sensory roots.

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23. Hassin, George B.: Tabes Dorsalis, Pathology and Pathogenesis: A Preliminary Report, Arch. Neurol. & Psychiat. **21**:311 (Feb.) 1929.

This proliferation is secondary to an epidural inflammation. The process is thus primarily a mesothelial inflammation with secondary degeneration of the ectodermal nervous tissue from obliteration of the spaces. This helps to explain the early reduction of the vestibular reactions to turning, for the vestibular nerve is one of the most primitive of the sensory nervous systems. In syphilis, deafness is a later manifestation, probably because of the greater protection afforded the nerve in the arachnoid prolongation surrounding the internal auditory meatus; although Richler said that toxicity of the cerebrospinal fluid is an important factor in the production of tabetic changes in the cord (Ingvar), which would support the view that the changes are caused by toxicity in the cerebrospinal fluid following syphilis, chronic infections or hemorrhagic trauma, which cause the reduction of the duration of the induced nystagmus to turning.

IMPAIRED HEARING AND MENINGEAL SYMPTOMS IN ANEURYSMS OF THE BASE WITH SUBARACHNOID HEMORRHAGE

Shore [24] stated that deafness in patients with basilar aneurysms has repeatedly been reported, although the lesion is some distance from the eighth nerve. His conclusions are: 1. Microscopic, true, arterial intracranial aneurysms are found in about 1 per cent of the brains examined at autopsy. 2. They are usually single and are seen most often on the basilar, internal carotid and middle cerebral arteries. 3. Syphilis as the cause of intracranial aneurysms usually manifests itself in the vertebral and basilar arteries, and as a factor in the sum total of intracranial aneurysms it plays only a small rôle. 4. The majority of intracranial aneurysms showing definite pathologic lesions are due to arteriosclerosis, while others with less definite lesions have been ascribed to infected emboli and a congenital weakness in the vessel walls. 5. Repeated hemorrhages from aneurysms at the base of the brain give rise to the most characteristic clinical symptoms of acute meningitis, viz., stiff neck, high temperature and headache, but a diagnosis of intracranial aneurysm is rarely warranted.

In my experience (1) hemorrhages into the arachnoid spaces of the base are common in infective diseases as well as in aneurysms. They give rise to (2) symptoms of meningitis, that is, high temperature, stiff neck and severe headache. Puncture of the spine gives a straw-colored fluid which is sterile, and this establishes the diagnosis. It is also my experience that many patients with aneurysms of the base (3) have a loud blowing noise in their head at the time of the bleeding but (4) knowledge of this is obtained only by questioning the patient, because the violent headache engages all his attention.

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24. Shore, B. R.: Intracranial Aneurysms, Arch. Path. **6**:181 (Aug.) 1928.

VESTIBULAR REACTIONS IN TRAUMA

Osnato and Giliberti [25] reported on 100 cases of postconcussion neurosis which they would call traumatic encephalitis; the symptoms of which they contrasted with epidemic encephalitis (lethargica). Nineteen per cent of the patients with concussion complained of tinnitus, whereas none of the patients who had epidemic encephalitis (lethargica) did so. Thirty-two per cent of the patients with concussion complained of defective hearing. It is notorious how frequently the mechanisms of the middle and internal ear are disturbed with injuries of the skull, and it is also recognized that the eighth pair of nerves is not commonly affected in cases of epidemic encephalitis.

Among the twelve cases of defective hearing caused by fracture of the base, five had vestibular disturbances. Headache in cases of concussion is almost invariably accompanied by dizziness, a symptom which was present in 57 of the 100 cases.

Osnato and Giliberti would confine the term dizziness to those cases in which an actual vertigo is present, and giddiness to the subjective feelings of unsteadiness connected with subjective visual disturbances—blurred perception of moving objects or clouded vision. The presence of a true vertigo was confirmed in a great many of their cases by disturbances of gait, the presence of nystagmus on changes of posture, or some abnormalities of the after-turning Bárány reactions. These authors say that I had stated that a syndrome of concussion of the brain or of increased intracranial pressure on the posterior fossa has been established. This I had described as reduction in the duration of nystagmus after stimulation of the horizontal canals, by turning with absence of or marked reduction in the response from the vertical canals to the cold caloric; they repeat my statement that the vestibular manifestation of increased intracranial pressure—absence of reactibility of the vertical canals of both ears to the cold caloric test—is of the greatest value in the early diagnosis of increased intracranial pressure as it appears long before and independently of papilledema.

Although Osnato and Giliberti found that the responses to turning and caloric tests were varied, yet there were in some cases definite indications of abnormality. In the caloric reactions, when the vertical canals were tested, the response either was absent as far as nystagmus went, or was prolonged or normal as to duration, but attended by excessive vertigo.

Osnato and Giliberti were unable to make out any definite syndrome of concussion based on the chair-turning and caloric tests, but they

25. Osnato, Michael, and Giliberti, Vincent: Postconcussion Neurosis; Traumatic Encephalitis: A Conception of Postconcussion Phenomena, Arch. Neurol. & Psychiat. **18**:181 (Aug.) 1927.

expressed the belief that dizziness on change of posture with nystagmus in any of the planes seems to point to injury of the vestibulocerebellar pathways.

They stated that perhaps the most characteristic feature of the Bárány tests in concussion is a tendency to a prolongation of the after-turning nystagmus, but more particularly excessive vertigo.

I no longer accept the dictum that the vertical and horizontal canals are stimulated separately by the cold caloric test, although this was my original statement.[26]

In fact, physiology teaches otherwise. One stimulates in the horizontal and vertical positions of the patient's head, not simply his horizontal and vertical canals, because during the stimulation all canals are affected. It was this effort—viz., to make the facts fit a theory—that would reduce living nervous tissue to the rôle of telegraph wires that is accountable for some of the discredit in which the vestibular tests are now held. Extended experience, however, confirms my original observation; viz., with increased intracranial pressure there is a marked reduction of irritability to the cold caloric test in the upright position, while it is present in the horizontal position. This is most strikingly seen in tumors of the acoustic nerve and in fractures through the labyrinth, because in these cases all hearing and caloric reactions are absent on one side and the pressure reaction is thus uncomplicated; but it is also present in a modified form in any increased intracranial pressure such as in acute brain abscess. With a continuation of the pressure the factors become more confused because of the development of compensation. Turning after injury or inflammation to the posterior fossa with hemorrhage or exudate in the arachnoid mesh gives a reduction of 50 per cent in the induced nystagmus, which in my experience lasts for a long time. This reduction, while of no localizing value, is of definite assistance in the diagnosis of a cerebral injury in these cases in which the patients claim compensation on account of vague headache and dizziness without other neurologic signs.[27]

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26. Eagleton, Wells P.: Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin: Report of Three Cases with a Previously Undescribed Aural Condition, Tr. Am. Otol. Soc. **13**:564, 1912; Laryngoscope **22**:592, 1913.

27. Eagleton, Wells P.: Vestibular Tests in Intracranial Surgery, Tr. Am. Laryng. Rhin. & Otol. Soc., 1922. This article shows the importance (1) of the absence of reactibility of the vertical canals of both ears to stimulation by the cold caloric test in the diagnosis of increased intracranial pressure of the posterior fossa, and (2) of the reduction of the duration of the nystagmus from turning in diffuse lesions of the cerebrospinal system pathways over the cerebellar cortex (as in protective meningitis and linear fractures of the skull of the posterior fossa).

THE ASSOCIATION OF FOCAL INFECTION (CAUSING MILD INTERMITTENT BLOOD STREAM INFECTION) WITH TRAUMA IN THE PRODUCTION OF EPIDURAL ABSCESS OF THE SPINE

During the past year I have seen a case of rarefying osteomyelitis of the vertebrae (which did not go on to suppuration) with chills and fever and the subsidence of all symptoms after the extraction of several teeth with apical abscesses.

Similar lesions have followed nasal and tonsillar disease. Hassin [28] reported a case of circumscribed suppurative (nontuberculous) peripachymeningitis with a histopathologic study, which is of interest to the otologist especially as it describes the pathologic process which is present in all epidural abscesses.

A young girl presented furuncles of the face, neck and buttocks of one year's duration. In December, 1925, she had furuncles on the face and back of the neck. Examination of the blood revealed 84 mg. of sugar per hundred cubic centimeters. Cultures from the furuncles showed a predominance of *Staphylococcus aureus*. In August, 1926, she again began to have "boils," one on the chest, two around the rectum, and on August 19, another in the lower thoracic portion of the back.

On August 22, she assisted for a period of forty-five minutes in administering artificial respiration to a girl who drowned, and she "thought she sprained her back," following which she experienced a sharp pain in that region. About four days after the physical exertion, she complained of severe pain in the back and chest, and on September 1, she felt numbness in the lower extremities and had difficulty in moving them. The same night she was paralyzed and unable to void urine or to have a bowel movement.

Examination showed a complete paraplegia of the lower extremities, bilateral Babinski phenomenon and complete anesthesia up to the level of the twelfth thoracic segment, and pain over the middle portion of the spine and rectal and urinary retention. The breathing was at times dyspneic, but the temperature was normal and vasomotor and trophic disturbances were absent. Hassin (September 3) interpreted them as manifestations of an extradural compression of the spinal cord.

On September 7, the patient complained of a "desire to void." The nurse started to catheterize, and in so doing moved the patient's legs. Almost immediately the patient became pulseless, developed Cheyne-Stokes respiration, foamed at the mouth, and died within fifteen minutes.

Necropsy revealed in the eighth and ninth vertebrae, between the periosteum and the posterolateral surface of the dura, a thick mass of granulation tissue in which were enclosed three small abscesses. The mass was loosely attached to the periosteum and was adherent to the dura. *Staphylococcus aureus* was isolated from all these abscesses.

Microscopic examination revealed three distinct abscesses in the form of dense foci of cell bodies; some were plasma cells, or lymphocytes; the majority were polymorphonuclear cells, many of which were enclosed within macrophages. The latter were numerous at the periphery of the abscess, where, in addition, many fibroblasts were present. The cell bodies—fibroblasts, macrophages, lympho-

28. Hassin, George B.: Circumscribed Suppurative (Nontuberculous) Peripachymeningitis, Arch. Neurol. & Psychiat. **20**:110 (July) 1928.

cytes, plasma cells and polymorphonuclear cells—formed a thick rim around the abscess—an embryonic capsule.

In the epidural mass bordering on the abscess capsule was a distinct connective tissue layer, unusually vascular and consisting of fibroblasts and a few polymorphonuclear cells. In some parts of the wall of the abscess, the blood vessels were numerous, hyperemic and infiltrated, with adventitial and endothelial cells, causing in some instances, obliteration of the lumen.

Aside from the three purulent foci with rather ill defined, incompletely formed capsules, there were other foci that possessed a connective tissue membrane much better developed. The contents of such foci were polyblasts or large lymphocytes, many of which showed mitotic figures. Plasma cells were exceptional here, while polymorphonuclear cells and macrophages were entirely absent. Hassin expressed his belief that such well encapsulated foci, with no polymorphonuclear cells, may be looked on as areas in which abscess formation was checked by a timely connective tissue proliferation. The microscopic field at this point appeared, therefore, to consist of granulation tissue and connective tissue strands that seemed to break up the former into numerous small foci, some of which were abscesses.

The dura generally formed a barrier against an extension of the inflammatory process. In many places it was directly invaded by the inflammatory elements. In such places, it appeared thickened and blended with the epidural mass.

Hassin stated that the strain to the back undoubtedly played some rôle in the causation of the disease. The primary factor unquestionably was a hematogenous staphylococcus infection from an extensive furunculosis. This has been mentioned in a number of cases as the principal etiologic factor of diffuse and circumscribed epidural suppurations. Hassin stated that similar epidural cordal lesions have followed nasal and tonsillar disease.

ADVANCES IN INTRADURAL SURGICAL TECHNIC

Cushing and Bovie [29] reported on electrosurgery as an aid to the removal of intracranial tumors; Bovie having devised a new surgical unit in which no use is made of electrically heated wires or scalpels, the effects depending entirely on the heat developed by the current in passing from the active electrode into the tissues, or on the ohmic heat developed by the current in passing through the tissues. For certain surgical purposes (biopsy) it is possible (as in the procedure termed "fulguration") to dispense with the indifferent electrode entirely, no complete electrical circuit being made between the two terminals of the machine. Under such circumstances, the patient's body becomes charged and discharged with each alternation of the current; and with the high frequencies used, the amount of the current passing from the active electrode into and out of the tissues is sufficient to produce the desired effects. The physical principles involved in the Bovie method are complicated, since the currents used change their direction of flow

29. Cushing, H., and Bovie, W. T.: Electro-Surgery as an Aid to the Removal of Intracranial Tumors, Surg. Gynec. Obst. **47**:751 (Dec.) 1928.

from 1,000,000 to 3,000,000 times per second and distribute themselves in the electrical circuits in a manner very different from that of steady currents or alternating currents of far lower frequency, such as those employed in a lighting circuit. Thus the relations between voltage and amperage which hold for steady currents and for alternating currents of lower frequency are upset by what the physicist terms phase shifts.

There are three distinct ways in which the various high frequency currents delivered by the Bovie unit are used in electrosurgery: (1) superficial dehydration; (2) cutting, and (3) tissue coagulation.

1. For superficial dehydration the active electrode is held not in actual contact with but sufficiently close to the tissues to be dehydrated, so that sparks are sprayed across the intervening space. The very high temperature of the sparks is sufficient completely to dehydrate a thin outer layer of tissue.

2. For cutting purposes, the active electrode is energized by a different type of current. The cutting is not done by the electrode, which has no sharpened edge, but actually by the current which forms an electrical arc ahead of the electrode which by vitalizing the tissues separates them as though they were cut. Further, by a suitable modification (variable amount of damping) of the same current a greater or lesser degree of coagulation or dehydration may be produced at will at the edges of the severed tissues. When the tissues are not particularly vascular the current is so modified as to produce the minimum amount of dehydration, but when the blood supply is greater the amount of dehydration is correspondingly increased.

3. For heating the tissues en masse (the so-called electrocoagulation), the kind of current used differs from that employed for cutting. An electrode energized with coagulating currents instead of having cutting properties cannot be moved through the tissues. The tissues surrounding the electrode become heated to a depth depending on two factors: the density of the current and the length of time it is permitted to flow. If a large tissue mass is to be coagulated, a comparatively weak current must be used for a prolonged period until it becomes heated throughout; for should a stronger current be used, the tissues in the immediate vicinity of the active electrode would become so quickly dehydrated or even carbonized that the current would cease to flow and the deeper tissues would not be affected. If, on the other hand, a small tissue mass is to be coagulated, one uses a strong current for a brief length of time, so that surrounding tissues are not affected.

I have used this unit to great advantage in the control of hemorrhage especially during the complete exposure of an extensive superficial abscess of the subdural space, a condition in which hemorrhage is not only embarrassing but frequently results in a failure of adequate dural incision.

IV. INTRADURAL SUPPURATIVE DISEASES THE RESULT OF OTITIS OR SINUSITIS

Diamond [30] reported the changes in the brain in two cases of pyemia and septicemia, one of *Staphylococci pyogenes* infection from chronic

30. Diamond, I. B.: Changes in Brain in Pyemia and in Septicemia, Arch. Neurol. & Psychiat. **20**:524 (Sept.) 1928.

otitis media and an acute osteomyelitis of the tibia with metastatic foci in many organs. The microscopic changes were mainly inflammatory with marked infiltration and hyperplastic phenomena in the pia-arachnoid, choroid plexus and glia, associated with the presence of *Staphylococcus pyogenes-aureus.* There were miliary abscesses near and in the vessels. The reactions in the pia-arachnoid and choroid plexus were polymorphonuclear cells, proliferation of the endothelial and adventitial cells and new formation of the capillaries. The glial reactions were granulomas and nodules, probably a reaction against a local infection the same as occurs in trichinosis. Besides those caused by the micro-organisms themselves there were many pathologic changes which Diamond believed were due to intoxication, as he stated that the hemorrhages which occur in the pia from acute sepsis are without micro-organisms. From certain strains of staphylococci Parker has isolated an exotoxin which when injected into mammals causes edema and infiltration with polymorphonuclear and mononuclear cells, followed by inflammation, necrosis and nuclear fragmentation of cells. This is the main point of Diamond's papers, as his second patient died, he thinks, chiefly of such cerebral intoxication.

The disease originated from extraction of teeth, and was associated with swelling of the face, numbness and pain, chills, high temperature and sweating. The patient became unconscious with eyes and head turned to the right; the pupils were dilated; there were nystagmoid movements and no reflexes; the spinal fluid was clear, under increased pressure, and contained 18 cells per cubic millimeter; the Wassermann reaction was negative.

Autopsy showed a large infarct in the spleen with diphtheric bacillus. The brain had minute areas of softening and thickening of the pia.

Microscopically, the brain showed two types of foci. One formed nodules; they were chiefly in the internal capsule and parietal lobe. In them the blood vessels were mostly empty and some larger vessels appeared thrombosed. The vessel walls showed marked signs of proliferation. The other type of foci resembled granulomas, but differed from the true nodules in the presence of many newly formed capillaries with areas of softening in the process of healing.

Diamond said that in his first case the foci of infiltration were caused by direct invasion of the parenchyma by the micro-organisms, viz., pyemia. But in his second case, the reactive phenomena—multiple foci of softening—were the result of the invasion of the parenchyma by toxins which produced septicemia without relation to the blood supply.

He drew the general conclusions that (*a*) infiltration phenomena denote the presence of an active virus in the form of a micro-organism and that (*b*) nonmicrobic or toxic factors do not produce infiltration but productive phenomena such as is seen in lead encephalitis.

PATHOLOGY OF CEREBRAL SUPPURATION

Atkinson [31] stated that the blood supply of the cerebral tissue comes from the circle of Willis by two types of vessel: (1) central branches, which pass directly into the base of the brain to supply the central nuclei and the main mass of white matter; (2) cortical branches, which pass over the surface and into the fissures. The latter ultimately divide into two varieties of terminal branches: (*a*) a large number of short vessels which supply the gray matter of the cortex only, and (*b*) a smaller number of longer branches which pass through the cortex to supply a thin layer of subadjacent white matter. These are all end-arteries, with no anastomosis between the central and cortical branches, so that there lies, just below the cortex and between the terminations of the cortical and central vessels, a flat area of white matter which is a relatively avascular zone. This avascular zone is present in both the cerebrum and the cerebellum, but owing to the greater complexity of the cerebellar convolutions its position corresponds to the center of the white core of each cerebellar folium which is supplied from either side by the cortical branches, the central vessels supplying the central white matter only. It is in this avascular zone, owing to the poor blood supply, that brain abscesses originate.

Infection is carried to this zone by the perivascular spaces from the subarachnoid space by a reversal of the normal flow. These two facts, the presence of an avascular zone and the reversed flow of cerebrospinal fluid in the perivascular spaces, form the basis of the pathology of adjacent abscess.

Adjacent Brain Abscess.—In adjacent brain abscess the original infection gradually spreads until it reaches the dura, causing a pachymeningitis, with or without the presence of an extradural abscess. This area of pachymeningitis induces a subjacent leptomeningitis, so that adhesions are formed between the membranes and the surface of the brain, shutting off the infected area from the main subarachnoid space. From this area of localized meningitis, infection passes into the brain substance by one of three routes—perivascular, venous or arterial.

The Perivascular Route: Infection by the perivascular route was originally put forward as a possibility by Macewen, and despite statements to the contrary (Eagleton),[32] Atkinson stated that it is by far the most common, as evidence of it was found in thirteen of sixteen specimens (82 per cent) examined by him. Sections of the earliest stage show the pial vessels to be patent, but their surrounding perivascular sheath distended by an infiltration of inflammatory cells, mainly

31. Atkinson, E. Miles: Abscess of the Brain: Its Pathology, Diagnosis and Treatment, Lancet **1**:483 (March 10) 1928.

32. Eagleton, W. P.: Brain Abscess, New York, The Macmillan Company, 1922, p. 18.

mononuclear, adherent to the cortex. This infiltration passes into the fissure against the normal stream of cerebrospinal fluid, owing to the local increase of pressure. The attack on the brain is extremely localized, as evidenced by edema and new vessel formation, occurring in one lobule only.

The Venous Route: Infection by the venous route was found only in two of sixteen specimens (12 per cent). When present, the lobule drained by the veins running into the thrombosed pial vessels is degenerating from interference with its blood supply. This interference is sufficient to cause changes even in the well nourished cortex, but produces its maximum effect at the point of least vascularity in the avascular zone. Thus again the development of the abscess is in the center of the white core of the lobule in precisely the same position as that in which the abscess originated by the perivascular route. One characteristic feature of thrombotic cases is the presence of scattered patches of hemorrhage which occur around the abscess, owing to the rupture of small capillaries from overdistention.

The abscess once formed extends in a different direction from those of perivascular origin. In the latter, the cortex is preserved intact owing to its good blood supply, the abscess extending backward through the center of the infected lobule. When the infection is by the venous route, however, the cortex is damaged and extension follows the course of the thrombosis; that is to say, outward toward the surface, and in doing so destroys the cortex. In these cases the abscess is therefore more likely to track to the surface and burst into the subarachnoid space, causing a generalized meningitis.

The Arterial Route: Atkinson found the arterial route to be the rarest of the three. It occurred in only one of sixteen specimens (6 per cent).

Capsule: According to Atkinson, the use of the word capsule is unfortunate, as it has served only to produce confusion of thought as to the real nature of the limiting membrane. It is in no sense a true capsule as applied to the tissue surrounding a tumor. It is nothing more than the ordinary wall of an abscess cavity, such as occurs in any part of the body, differing in histologic structure only so far as the supporting tissue of the brain differs from that in other parts. In the chronic cases with infection of low virulence, the wall of the cavity will be found to consist mainly of fibrous tissue of greater or lesser thickness, with a consequent variability in the degree of neuroglial hyperplasia. Internal to this is a layer of characteristic cerebral granulation tissue; viz., capillaries contained in a supporting tissue of endothelial cells, leukocytes, neuroglial cells and fibroblasts—while external to the fibrous tissue layer is an encephalitis of variable degree and extent.

In the acute abscess, the result either of a vascular catastrophe or of a fulminating infection, there has been no time for the development of any protective mechanism, and there is no evidence of a limiting pyogenic membrane. The contents of the cavity consist of necrotic brain tissue, and a wall of acutely inflamed cerebral tissue. It is rather an acute spreading encephalitis with tissue necrosis than a true abscess.

Direction of Spread: As the abscess gets larger it extends in a definite direction. In the cerebrum it increases at the expense of the white matter; the cortex is protected by reason of its good blood supply. In the main, extension follows the course of the vessels inward toward the lateral ventricle. The more acute the process the more likely is it to perforate the ventricular wall, whereas in chronic cases the choroid plexus tends to become adherent in the region of threatened perforation, acting in the same way as the omentum in the peritoneal cavity. In the cerebellum extension also occurs at the expense of the white matter, but the central mass at first is involved only to a minor degree. The process starts from the anterior end of the cerebellum, picks out one particular lobule and spreads backward through that lobule. Adjacent lobules are involved only as the size of the abscess increases. The central mass of white matter is not usually destroyed to any great extent and the abscess itself generally does not approach the central cerebellar nuclei, in particular the dentate; hence localizing signs will be absent unless these nuclei are involved by the surrounding encephalitis. The actual abscess is usually ovoid with smooth walls.

Infection of the cerebellum enters its substance by way of one of the main fissures, usually either the great horizontal or the fissure below. This is because the saccus endolymphaticus lies in relation to the inferior lip of the great horizontal fissure, while the knee of the lateral sinus lies actually on the fissure and turns downward over the postero-inferior lobule. These being the two common routes of infection, the postero-inferior lobule is the one most likely to be involved by the cerebellar abscess.

The Direction of Exploration: In the cerebrum, the direction of exploration, if one is working from the known point of entry, should be toward the ventricle.

In the cerebellum, the direction of blind exploration, if one is working from the anterior aspect, should be backward and upward or downward, that is, on either side of the great horizontal fissure.

Davidson's Case.—Davidson [33] reported the case of a patient with brain abscess of many months' duration which ultimately caused death by cerebral hemorrhage, a condition which I have encountered but once.

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33. Davidson, Charles: Brain Abscess Following a Pyemia of Long Duration, Tr. N. Y. Acad. Med., Sect. Neurol. & Psychiat., April 9, 1929.

A patient immediately following an abortion developed pelvic peritonitis followed by a pyemia. Five years later a retro-orbital abscess formed which was drained, followed by a brain abscess localized in the frontal lobes. After drainage meningitis and hemiplegia with hemianesthesia ensued. The meningitis disappeared but the paralysis and sensory disturbances remained. Two years later the patient died suddenly, the ultimate cause of death being a subarachnoid hemorrhage. This hemorrhage evidently was caused by damage to the vessel walls by the infection.

Symptoms of Brain Abscess.—Symonds [34] said that in cerebral abscess, during the early stages the signs of increased intracranial pressure are limited to occasional headache often experienced only on walking, and increased by stooping and coughing. Severe headache, vomiting, drowsiness and slowing of the pulse probably mark the occurrence of mechanical obstruction to the outflow from the ventricles with internal hydrocephalus.

Aphasia.—A patient may be suffering from a definite aphasia and yet be able to converse without apparent difficulty.

In the case of a boy, aged 16, the house surgeon stated that there was no aphasia. A collection of heterogeneous objects was taken, and the patient was asked to name them. Books, matches, pen, watch and half a dozen other articles were named correctly. There were only two failures. When shown a key he could describe its use but could not name it. He rejected all substitutes and promptly accepted the word "key" as correct. Also when shown a plate, he said it was to eat off, made of china, but could not name it. As before, he rejected substitutes and accepted the correct name when it was given to him.

The ease with which aphasia may be overlooked is emphasized by Sonntag,[35] He collected from the literature the reports of 177 cases of left-sided temporal lobe abscesses in which examination was made for aphasia; there were speech disturbances in 155 of the cases (87.5 per cent). Of twenty-two supposedly normal patients, only three had been properly examined. Sonntag said that in right-handed persons, a left-sided temporal lobe abscess very rarely runs its course without aphasia. When motor aphasia is present with the sensory aphasia, it is usually referable to meningitis (nine cases). Verbal amnesia was observed 101 times. In forty cases, paraphasia was present.

The extent of the foci has no notable influence on the aphasia.

In a previously mentioned article,[11] I called attention to the value of the diagnosis of edema of the brain in cases of cerebellar and cerebral abscess. I pointed out that:

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34. Symonds, C. P.: Diagnosis and Localization of Cerebral Abscess, Proc. Roy. Soc. Med., Sect. Otol., 1928, p. 1139.

35. Sonntag, R.: Contribution to the Knowledge of Speech Disturbances in Otogenic Temporal Lobe Abscesses, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:434, 1927-1928.

1. Routine daily recording of visual fields by a trained technician will frequently add localizing information in suppurative diseases within the dura.

2. Edema of the brain, the result of suppuration, can be diagnosed, if the edema involves the optic tract, by uneven hemianopic indentations of the contralateral side. These indentations frequently are transient.

3. The edematous nature of the lesion is shown by constant variations in the fields.

4. Cerebral edema may extend from the posterior fossa to the great brain, although a localized abscess is confined to the posterior fossa itself. Such fields are generally contracted, as well as having a general hemianopic tendency.

5. Localized bulbar cisterna meningitis may be diagnosed by a combination of middle and posterior fossa symptoms, associated with bitemporal hemianopic identations and a peculiar type of semicoma.

6. Papilledema in suppurative lesions possesses some localizing value.

7. Fourth nerve paralysis localizes the lesion in the posterior fossa near the median line. Third nerve paralysis has little localizing value.

8. Sixth nerve paralysis stands between the two in localizing importance.

9. Pain behind the eye from irritation of the ophthalmic branch of the fifth nerve places the lesion in the middle fossa.

10. Dysfunction of the oculomotor apparatus may be tested by the vestibular tests through which (*a*) increased intracranial pressure and (*b*) effusions over the cerebellar cortex may be demonstrated.

Unusual Types of Brain Abscess.—Federici [36] reported six cases of temporosphenoidal brain abscess and two cases of cerebellar abscess. In seven cases, the streptococcus was found (four times, *Streptococcus mucosus*); once, the colon bacillus. Contralateral deafness was noted in one instance. In one case of left-sided temporal lobe abscess, amnesic aphasia was absent. The cerebellar abscesses were always accompanied by vertigo, vomiting and spontaneous nystagmus toward the affected side. The pointing test gave negative results. The abscess was double in the cerebellum in both cases.

Bernardie's [37] case illustrates the frequent improvement which follows an operation on the ear in the presence of an intradural collection of pus, and the danger of allowing a patient out of bed after an apparent

36. Federici, F.: Some Cases of Cerebral Abscess of Otitic Origin, Ann. di laring., otol. **29**:166 and 189, 1928.

37. Bernardie, C. B.: Brain Abscess of Otitic Origin, Rev. de laryng. **49**:620, 1928.

cure of a brain abscess. I have gradually lengthened the time until now I try to keep my patients who are recovering from cerebral abscess confined to the bed for from two to three months.

Bernardie's patient was admitted to the hospital with acute pressure symptoms, rigidity at the nape of the neck, etc. Lumbar puncture showed increased pressure and turbidity. Mastoidectomy was performed on the next day, with subsidence of all meningeal symptoms. Daily punctures yielded a progressively clearer fluid. On the fifth day, the rigidity of the neck had disappeared, the temperature was normal and the condition was regarded as aseptic purulent meningitis. On the ninth day after the operation, the patient again had severe headache; no Kernig sign, rigidity of the neck, or adiadokokinesis was noted; the pulse rate was 68. On the following day, he had total aphasia. Incision of an abscess was done. Immediately afterward, the patient spoke spontaneously. Nearly three weeks later, when he was apparently well, meningitis supervened. Autopsy showed pus at the base. In the temporosphenoidal lobe there was a cavity free from pus. There was no encephalitis in the vicinity.

Brock [38] reported twenty-six cases of brain abscess; in fourteen the abscess was in the temporal lobe, and in twelve in the cerebellum. In three of the cases autopsy reports were given. The dura was intact in only 17 per cent of twenty-four cases. It was thickened and had granulation on it in six cases and was discolored in thirteen cases. A fistulous tract was present in six cases. Only three of the fourteen cerebral abscesses and only one of the twelve cerebellar abscesses were uncomplicated. Brock reported recovery in 41 per cent of his cases.

In the diagnosis Brock attaches value to the presence of obstinate constipation which resists massage, laxatives and enemas. He said that the absence of constipation is presumptive evidence against the existence of a brain abscess. Isolated rigidity at the nape of the neck in the absence of the Kernig sign is also useful.

Giannetto [39] reported a case of "Latent Cerebral Abscess of Otitic Origin," which is of interest as showing for how long a time (a month and a half) a cerebral abscess may be in existence with scanty symptomatology. Bacteriologic examination of the pus showed the presence of gram-positive diplococci (pneumococcus type).

Bryan [40] reported a case in which abscess was found in the lower portion of the temporal lobe at operation. The patient died on the fourth day. Section of the brain showed a small cavity lying in the extreme posterior portion of the temporal lobe. The lateral ventricle was filled with pus, débris and necrotic tissue. He gave good illustrations of the abscess in the lateral ventricle.

38. Brock, W.: Experiences with Otitic Brain Abscess, Arch. f. Ohren-, Nasen-, u. Kehlkopfh. **118**:161, 1928.

39. Giannetto, J.: Latent Cerebral Abscess of Otitic Origin, Riv. oto-neuro-oftal. **3**:481, 1929.

40. Bryan, J. H.: Abscess of the Brain of Otitic Origin, Ann. Otol. **37**:819, 1928.

V. SUBDURAL SPACE ABSCESS

Circumscribed pachymeningitis interna purulenta is of very rare occurrence. Blegvad found only twenty-four cases in the literature. The subdural abscess in the majority of the cases was associated with another intracranial complication. The prognosis of subdural abscesses is relatively favorable, provided there is no other intracranial complication. In Revesj's [41] case a pachymeningitis purulenta interna led to the formation of a subdural abscess. There was pus under the dura above the horizontal semicircular canal. The dura did not pulsate. After removal of the tegmen a fistula leading under the dura was plainly visible. A crosswise incision was made in the dura.

Jakub [42] reported a case showing how extensively pus in the subdural space will travel.

A patient with right-sided otitis media, had exophthalmus, choked disk and impaired motility of the eye on the opposite side. Right-sided radical operation showed an intact dura and normal sinus; brain puncture yielded no pus. Later an opening was made above the left eye; some pus was voided on puncture. Autopsy revealed the left (opposite) hemisphere covered with pus, which was entirely subdural.

Borries [43] emphasized the differences between diffuse meningitis and protective (Martland) meningitis from an abscess. The latter may be demonstrable only by microscopic examination. It is usually benign, is characterized by the lumbar fluid having slight cellular increase, and is sterile or has a gradual subsidence of the microbes. It is rare for the cerebrospinal fluid to be entirely without cytologic changes in brain abscess. Generally there exists a "minimal meningitis with a small number of cells, but sterile."

Symonds,[44] in reporting a case of "superficial brain abscess," said that the distinguishing features are the sudden onset of convulsions or paralysis of the cortical type and the changes in the cerebrospinal fluid. The convulsions may be falsely interpreted as the first sign of a generalized meningitis. As the process is slow and involves an area which in the neurologic sense produces few symptoms it may remain "silent" until it reaches the sylvian fissure.

A child with acute left otorrhea was admitted to the hospital. At the operation, the dura of the roof of the antrum was injured. On the eleventh day after opera-

41. Revesj, R.: Contributions to the Pathology and Therapy of Subdural Abscess, Gyógyászat **68**:122, 1928; Zentralbl. f. Hals-, Nasen- u. Ohrenh. **12**:422, 1928.

42. Jakub, L.: A Case of Contralateral Cerebral Complication in Chronic Purulent Otitis, Russk. Oto-Laring. **20**:612, 1927; Zentralbl. f. Hals-, Nasen- u. Ohrenh. **12**:272, 1928.

43. Borries, G. V. T.: Diagnosis of Cerebral and Subdural Abscesses by Lumbar Puncture, Ann. d. mal. de l'oreille **47**:452, 1928.

44. Symonds (footnote 34, p. 1142).

tion, right-sided jacksonian epilepsy developed, followed by right-sided clonic convulsions. Lumbar fluid, although clear, contained a slight excess of lymphocytes. The epileptic attacks suggested that the patient had a cortical, and therefore a superficial, lesion; the spinal fluid suggested that the infective focus was shut off from the general subarachnoid space. There was a small patch of granulations present on the dura over the roof of the antrum (the place where the dura was injured at the operation) in the center of which a bead of pus was seen. When the opening was enlarged, a quantity of pus escaped under considerable pressure.

It is my experience that abscess in the subdural space of otitic origin seldom occurs over the temporal lobe without there having previously been an operative or traumatic injury of the dura as in Symonds' case, this for the anatomic reason that the brain is held firmly to the dura in this region and consequently infection ordinarily passes directly through the subdural space. However, with an opening in the dura from the outside the infection may enter the subdural space and spread over the temporal lobe, sometimes for a great distance. Abscess in the subdural space is not an uncommon result of the evacuation of a temporal lobe abscess.

On the other hand, localized collections of pus in the subdural space are very common over the frontal lobe. My experience leads me to believe that they form probably 25 per cent of all the frontal lobe infections. Subdural space abscess also occurs over the anterior surface of the cerebellum; they are very limited in extent. In all locations they are diagnosable. They are of good prognostic import if thoroughly exposed by free incisions of the dura which must extend up to and beyond the termination of the infection. The full extent of the abscess may be very difficult to determine and is only possible by the sacrifice of extensive areas of bone.

TREATMENT OF BRAIN ABSCESS

Coleman [45] opposed the formation of a large osteoplastic flap as he had not found pus in the piarachnoid meshes. He expressed the belief that when a patient has signs of general intracranial pressure the abscess is probably subcortical. In two cases he used the flap because he then was unable to differentiate between abscess and tumor. He taps the abscess with a small needle and uses it for drainage. Death, he believes, is generally due to encephalitis from the trauma of the operation.

Coleman's experience in encapsulated abscess leads to the following conclusions:

1. There are a number of types of localized intracranial suppuration. Treatment suggested for one may be entirely unsatisfactory in another.
2. An encapsulated abscess offers a good prognosis if accurately local-

45. Coleman, C. C.: The Treatment of Abscess of the Brain, Arch. Surg. **18**:1 (Jan.) 1929.

ized and properly treated. 3. The cause of death following operations on abscesses is nearly always septic encephalitis brought on by efforts at drainage. There appears to be little danger of a fatality after partial collapse of the abscess, provided pus does not escape from the abscess into the surrounding brain tissue. 4. The author prefers to drain encapsulated abscess, but he expressed the belief that this should be done through a trephine opening made with the patient under local anesthesia. 5. It is important to approach the abscess at the point nearest to the cortex, but such a point cannot always be determined. He stated that simple tapping of an encapsulated abscess is to be preferred to drainage, if drainage is accomplished with difficulty or when many attempts are necessary to insert the drainage tube into the abscess cavity.

In fourteen of his cases of chronic abscess, he reported two deaths.

Potts'[46] five patients were all operated on according to the same technic, viz., exposure of the dura by an extension of the incision from the mastoid wound. The cerebellar abscesses are drained from below and back of the lateral sinus and the temporosphenoid by uncovering the dura above the ear. In each case bone is removed to an area of about 4 cm. A small incision is made through the dura. If the abscess is definitely located, the Gifford brain abscess seeker is passed into the abscess; if the localization is not definite, a large blunt needle with a cannula is used. The Gifford seeker is introduced by the side of the needle, and the needle is withdrawn.

Arsphenamine and Brain Abscess.—Heyninx[47] reported right-sided brain abscess due to chronic mastoiditis, treated by drainage with a rubber tube. As the bacteriologic examination showed spirilla and fusiform bacilli, neoarsphenamine was administered. The patient recovered.

At the Manchester meeting of the British Medical Society, 1929, Cohen of Liverpool stated that four cases of brain abscess of otitic origin gave a positive Wassermann reaction without a history or other evidences of syphilis.

After-Treatment of Brain Abscess.—Muck[48] expressed the belief that improved statistics depend on the development of the after-treatment rather than on improvement of the operative methods. He stated that it is advisable to place the patient in the sitting position after the evacuation of the abscess and at the same time to turn the head toward

46. Potts, J. B.: Localization and Drainage of Brain Abscesses of Otitic Origin, Ann. Otol. **32**:513, 1928.

47. Heyninx: Gangrenous Brain Abscess, Oto-rhino-laryngol. internat. **12**: 204, 1928.

48. Muck, O.: Comments on the Cause of Healing as Well as the Fatal Outcome in a Series of Cases of Brain Abscess, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:410, 1928.

the healthy side, so that the abscess cavity becomes dilated. Thus the formation of a recess is prevented. He has the patient maintain the seated position for from eight to fourteen days. He also employs irrigation, with the patient in the seated position. In cerebellar abscesses, a counter opening at the nape of the neck is recommended. Twelve otogenic and two rhinogenic brain abscesses were observed. Two cases were not recognized until the autopsy. Eight patients survived.

In a later article Muck gave the details of his treatment [49] in a specific case. Closure of the wound in the brain was obtained by: (1) thorough irrigation of the cavity, (2) placing the patient in a sitting position, (3) ventilation with the aid of the capsule dressing and (4) breathing gymnastics.

After incision of the brain, 30 cc. of pus and brain particles was evacuated. The patient was placed in a sitting position and the head was turned sharply to the right. A large cavity was unfolded which was not visible when the patient was lying down. After the protruding parts of the brain had been removed with the sharp spoon, the cavity was plainly visible.

The cavity was irrigated with 1 liter of hydrogen dioxide solution with the patient in a sitting position. A glass tube of the diameter of 1.5 cm. was placed in the cavity. This allowed a good view. The patient was kept in a sitting position for the following eight days.

As Muck had noticed the action of respiration on the size of the cavity, he used it methodically. The patient was ordered to take a deep breath seven times and then to hold her breath for a few seconds. This breathing procedure was carried out every hour during the first day and then every two hours.

Details of Progress from Day to Day: First day: The wide glass tube was completely free of fluid. After its removal, a circular opening remained in the brain which allowed a good inspection of the cavity. It was pear-shaped. The longitudinal diameter of the cavity was 5.3 cm., and the transverse diameter was about 3 cm. The cavity was irrigated with 1 liter of hydrogen dioxide solution. Small particles of brain appeared in the fluid. The cavity was illuminated with the heat emitting lamp for three minutes. The tube was reintroduced. Second day: The tube was not clogged. Some viscous fluid was found on its floor. The cavity was irrigated and illuminated with the heat emitting lamp. The tube was reinserted. Third day: There was very little fluid in the tube. The cavity was much smaller. The brain had advanced, especially from above. The cavity could be inspected easily. If the patient was placed in a horizontal position, the abscess cavity became so small that it closed up entirely in front of the drainage tube. Brain substance appeared before the opening of the tube. If the patient was again placed in a sitting position, the cavity unfolded itself and appeared again in its original form. Fourth day: The cavity was free from pus and had become still smaller. Fifth day: The glass drainage tube was shortened. A capsule made of aluminum, with holes, was placed in the region of the ear so that the air had access to the cavity in the brain. With this capsule dressing and with the patient in the upright position, the cavity was only the size of a pea. When

49. Muck, O.: Ventilation of Evacuated Abscess Cavities of the Brain, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:343 (Jan. 7) 1929; trans. by Dr. Emil Amberg.

the patient took seven deep breaths and held her breath for a few seconds, the cavity became larger, reaching the size of a small hazelnut. The cavity was free from fluid. Seventh day: The cavity did not contain any fluid. The heat emitting lamp was employed after seven deep breaths. Eighth day: While the patient was sitting, the cavity could not be seen. After seven deep inspirations, a cavity the size of a pea could be seen when the patient held her breath. Ninth day: Even after deep inspirations, the cavity could not be seen.

PNEUMO-ENCEPHALITIS WITH BRAIN ABSCESS

Uffenorde [50] reported a second case [51] of pneumo-encephalon in a temporal lobe abscess. The temporal lobe abscess was opened; the patient died fifteen days later. Autopsy showed rupture of a large abscess into the ventricle. Here, as in a previous case, the author noted a distinct hissing noise due to the escape of air during the change of dressings and separation of the incision. He believed that the aspiration of air into the abscess cavity and into the ventricle was caused by movements of the head, coughing and swallowing, during the drainage of pus from the abscess. Uffenorde recommended bitemporal radiography at the time of the first change of dressings, in order to demonstrate the onset of postoperative pneumo-encephalon. If air is absent, the extent of the abscess cavity and its communication with the ventricle may be ascertained by filling it with iodized sesame oil 40 per cent. Only 0.5 per cent iodized oil should be employed, and the roentgenograms should not be taken until the day after the injection, for it is only after some time has elapsed that the iodized oil is distributed through the influence of the pulsatory and respiratory movements of the brain.

I have injected air into a number of abscess cavities for the purpose of demonstrating their extent, and while the latter has failed, I have been impressed by the apparent stimulus and healing that has been caused by the air, a theory which Muck [48] is apparently trying to apply.

It is possible that Uffenorde's case was caused by an anaerobic bacillus infection as originally described by me.[52]

NONPURULENT ENCEPHALITIS, THE RESULT OF OTITIS

Voss' [53] observations were on two cases of recent toxic nonpurulent encephalitis, as a sequel of influenza otitis (one developing gradually

50. Uffenorde, W.: A Further Observation on Rupture into the Ventricle and Spontaneous Pneumo-Encephalon in the Roentgen Picture in Otogenic Temporal Lobe Abscess, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:577, 1927-1928.

51. Uffenorde, W.: Ventrikeleinbruch und spontanes Pneumencephalon im Röntgenbild bei einem Fall von otogenem Schlafenlappen Abscess mit Ausgang in Heilung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:567, 1927-1928.

52. Eagleton (footnote 32, p. 188).

53. Voss, C.: Otitis Media and Encephalitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:596, 1928.

without fever, and the other suddenly with fever) as well as on two cases of serous encephalitis, with slight symptoms of concomitant serous meningitis. These four cases led him to propose the following classification for the nonpurulent otogenic forms of encephalitis: 1. Serous encephalitis. This either remains (*a*) a serous meningoencephalitis or passes into (*b*) a serous encephalitis with (*c*) a serous meningitis. Serous encephalitis should be recognizable by the clinical history and the fluid changes. 2. Hemorrhagic encephalitis (apoplectiform, embolic form). 3. Toxic encephalitis (influenza) with a toxic influenzal origin, which because of the absence of autopsy observations must be different from either serous or hemorrhagic encephalitis.

Symonds,[44] speaking of "localized nonsuppurative encephalitis," stated that in the course of formation of every cerebral abscess there must be a presuppurative stage of inflammation with engorgement, exudation and swelling without necrosis. The inflammatory process may be resolved without pus formation. He stated, however, that it is doubtful whether such a localized nonsuppurative encephalitis occurs in relation to otitis media.

A boy had a right-sided otorrhea and mastoiditis, for which an operation was performed. Two or three weeks later, headache and drowsiness developed; slight swelling of both optic disks and some alteration of the reflexes on the left side of the body suggested a right temporal lobe lesion. An exploratory operation was performed but no abscess found. As the symptoms persisted a second operation was done ten days later. Again no abscess was found, but the boy subsequently made a complete recovery.

A second patient had left-sided otitis, headache and drowsiness. He was somewhat confused, was definitely aphasic and had a right homonymous hemianopia. He refused operation and subsequently was discharged from the hospital, having completely lost his aphasia and hemianopsia.

A third patient, a boy, was admitted to the hospital on account of pain in and behind the right ear following an acute otitis media of one week's duration. The temperature was 100 F. At operation, pus was found in the mastoid cells and a small collection compressing the lateral sinus which was not thrombosed. Following the operation, there was a swinging temperature between 99 and 105 F.; the pulse rate ranged from 90 to 120. Mentally, he was apathetic. On the nineteenth day after the operation, a doubtful extensor response was obtained from the left foot. He began to complain of right frontal headache, chiefly on waking in the morning. Twenty-five days after the operation he had a definite extensor plantar response on the left with diminished abdominal reflexes on the same side. A diagnosis of right temporal abscess was made. The dura was opened and the brain explored without any abscess being discovered. Lumbar puncture at the time of operation gave a clear fluid apparently under increased pressure, without increase of cells or protein. Six days after this second operation the physical signs were unchanged. The temperature and pulse rate were still high, and there was a considerable headache. He was given full doses of methenamine. Five weeks from the date of admission, he began to improve. He seemed normal ten days later and has continued well.

I would suggest that the pathologic lesion in the sudden apoplectiform cases was an infarct, involving a small cortical vein. The deposit of the infarct is accompanied by marked symptoms and followed by a protective meningeal reaction which may be pronounced. If the infarct is highly infective a local cortical abscess or a rapidly spreading meningitis will develop, but in a considerable proportion of cases it undergoes resolution. I have frequently encountered this lesion and have several pathologic specimens of it, one of which I exhibited at Manchester.

VI. FRONTAL LOBE ABSCESS

Szmurlo [54] emphasized that in spite of the relative frequency of nasal accessory sinuses—which occur in 34 per cent of all persons, according to Burger—intracranial complications are undoubtedly rare, much rarer than cerebral complications from the ear; the proportion of these two being 7:167 (Burger). I have endeavored to explain the rarity of intracranial invasion from accessory sinus suppuration on purely anatomicohistologic and physiologic relations. The majority are derived from frontal sinus inflammations. Statistical data show that ethmoidal frontal brain abscess represents hardly one tenth of all rhinogenic suppurations.

A noteworthy feature of Szmurlo's case was the insidious advance of the inflammatory process by the venous route.

The patient, a girl, had been ill for three weeks with frontal headache, right-sidered exophthalmos, with choked disk. There was fluctuation in the depth of the orbit. The suppurating frontal, ethmoid and sphenoid sinuses were opened and the orbital abscess evacuated. After ten days, trephining was done and about four spoonfuls of pus were evacuated from the right frontal lobe. Autopsy revealed a frontal brain suppuration from the ethmoid cells and the sphenoid sinuses.

VII. CEREBELLAR ABSCESS

NYSTAGMUS IN TRAUMATIC AND EXPERIMENTAL LESIONS OF THE CEREBELLUM

Ivy,[55] in speaking of the pathways of impulses causing labyrinthine nystagmus, stated that while lesions of the anterior portions of the cerebellum cause spontaneous nystagmus (which he believed is "ataxic") and that after the spontaneous nystagmus disappears the response to rotation is increased for months, however, labyrinth effects, including nystagmus, occur normally in the absence of the cerebellum, and destruction of the labyrinth does not prevent the appearance of the typical manifestations of cerebellar injuries. These facts upset the view that there is a definite pathway for nystagmus passing by way of the cerebellum.

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54. Szmurlo, J.: Frontal Brain Abscess as a Sequel of Purulent Ethmoid Cell Inflammation, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **13**:244, 1929.

55. Ivy, A. C.: The Physiology of Vestibular Nystagmus, Arch. Otolaryng. **9**:123 (Feb.) 1929.

As regards the cerebral origin of the quick component of vestibular nystagmus, Ivy said it certainly has not the integrity of a cerebral reflex arc. He expressed the belief that the quick component is dependent on some center below the thalamus. Over this center the cerebrum exerts an inhibitory influence.

NYSTAGMUS (VESTIBULAR REACTIONS) IN SUPPURATION

In central nystagmus [56] the patient may fall in the direction of the quick component of the nystagmus. This falling reaction is, as a rule, independent of the position of the head.

Disease of the nuclei of the labyrinth nerve produces either nystagmus to the diseased side, or nystagmus to both sides, more marked on the diseased side.

A combination of nystagmus and a paralysis of the recurrent laryngeal nerve may occur. It is anatomically explainable by the proximity in the medulla of the fibers of the vagus to the nuclei of the labyrinth nerve. Therefore, when these nuclei are affected it is usual for the vagus to become involved through extension of the process. Disease of the vagus fibers produces a paralysis of the recurrent laryngeal nerve on the diseased side. Thus, there is a syndrome (Brunner) which is characterized by a nystagmus to the diseased side and a "recurrent" paralysis on the diseased side.

Ferreri [57] reported a case of cerebellar pseudo-abscess due to otogenic serous meningitis in which the lumbar fluid was of a straw yellow color. A diagnosis of abscess was improbable because of the direction of the spontaneous nystagmus which was chiefly toward the diseased side with an intact labyrinth. The diagnosis of localized meningitis was based on the functional labyrinth examination, which showed a bilateral caloric hyperirritability with a "reversed nystagmus." The cerebellum showed no signs of increased pressure.

Russian authors [58] reported four cases of otogenic cerebellar abscess in which operation was performed; in two the abscess was found at the time of the operation. One patient recovered. In another case [59] importance is attached to the direction of the spontaneous nystagmus toward the side of the lesion, and to its great variability. All four cases showed spontaneous nystagmus to both sides. Neumann's symp-

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56. Brunner, Hans: Clinical Significance of Spontaneous Nystagmus, Arch. Otolaryng. **9**:1 (Jan.) 1929.

57. Ferreri, G.: Cerebellar Pseudo-Abscess Due to Otogenic Serous Meningitis, Riv. oto-neuro-oftal. **5**:247, 1928.

58. Markow, O., and Galperin, J.: Four Cases of Cerebellar Abscess, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **12**:774, 1928.

59. Canuyt; Terracol, and Klotz: A Case of Acute Cerebellar Abscess with Pathological and Therapeutic Comments, Arch. internat. de laryng. **7**:315, 1928.

tom (spontaneous nystagmus toward the diseased side on exclusion of the homolateral labyrinth) was present in four cases, and in the opinion of Markow and Galperin it is an indication for the performance of a cerebellar operation, even when all other cerebellar symptoms are absent. In the case that terminated in recovery, spontaneous nystagmus was still present sixteen months after the operation. Death occurred in each instance about three months after the onset, from paralysis of the respiratory centers.

INCOORDINATION, WEAKNESS AND PARALYSIS IN CEREBELLAR ABSCESS

Griess [60] stated that a practical clinical aid is not to confuse one-sided weakness, which accompanies incoordination, with a paralysis. In cerebellar abscess the incoordination is not proportionate to the weakness; the greater the weakness the more difficult it is to establish incoordination. Weakness occurs on the same side of the lesion and if associated with incoordination and involvement of the sixth nerve should cause the observer to suspect that the abscess is in the cerebellum.

CEREBELLAR ABSCESS BY WAY OF THE FACIAL CANAL

Cambrelin [61] reported a case of cerebellar abscess of otitic origin, and assumed that the transmission of the infection was by way of the facial canal, a very rare condition.

LATENT CEREBELLAR ABSCESS

Specht [62] reported a case of cerebellar abscess which was latent for several months after evacuation, as the patient enjoyed a period of good health. Sudden death occurred from meningitis.

In August, 1925, in the course of acute otitis media, the patient was attacked by severe pains in the forehead, with vomiting preceded by a sensation of vertigo. Antrotomy was followed by a period of improvement. Four weeks later severe pains were felt in the forehead and occiput. Surgical intervention failed to reveal any pathologic change of the dura over the cerebellum or in the posterior surface of the pyramid. After operation, the patient remained well for some months, and then again suffered from frontal headache, a vertiginous sensation (no rotatory vertigo) and vomiting. The pressure of the cerebrospinal fluid was slightly raised with a small increase of cells; the Wassermann and Pandy tests gave a negative reaction. The right cerebellar hemisphere was punctured with negative result. The patient was again immediately relieved. Two months later he again complained of severe frontal headache. There was no vomiting, vertigo or nystagmus, but

60. Griess, Walter: Surgery in a Children's Hospital: Its Status and Some Clinical Observations, Arch. Pediat. **45**:285 (May) 1928.

61. Cambrelin: Cerebellar Abscess of Otitic Origin, Rev. oto-neuro-opht. **6**:417, 1928.

62. Specht, F.: Contribution to the Question of Cerebellar Abscess Without Cerebellar Symptoms, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **120**:23, 1929.

he was extremely restless and excited. Psychiatric examination suggested a brain tumor. At operation, pus was aspirated at a depth of 6 cm. The large abscess was incised close to the vermis; from it two tracts passed in an anterior and lateral direction toward the lateral sinus. The brain abscess cavity closed in about three weeks by granulation. The patient was discharged in good condition.

Five months after the operation, cerebellar signs returned for a few days and again subsided. At the end of three months, meningitis supervened, and the patient died suddenly. Postmortem examination showed an enormous cavity in the cerebellum filled with viscid greenish-yellow pus.

Specht recommended, in the drainage of cerebellar abscess, that the operative area selected be behind the sinus as far laterally as possible. I recommended a lateral operation in spite of the greater tendency to cerebellar prolapse; I frequently enter through the obliterated sinus.

CEREBELLAR ABSCESS OF CONTRALATERAL SIDE FROM THE OTITIS

The statistics of the Milan Ear Clinic [63] comprise 103 cases of sinus thrombophlebitis. In one case the thrombophlebitis was associated with cerebral abscess, and in two cases with a cerebellar abscess. In one, symptoms of meningitis developed after an operation on an infected thrombosis of the sigmoid sinus. Autopsy showed an abscess on the contralateral half of the cerebellum.

CEREBELLAR ABSCESS FROM ACTINOMYCOSIS

Rockemer and Wirth [64] reported a case of chronic middle ear suppuration and cerebellar abscess from actinomycotic infection but without the formation of actinomyces granules. The fungi apparently did not belong to any of the varieties of actinomyces which have been described. They had morphologic and cultural differences from the actinomyces fungi with granule formation. The autopsy showed suppurative degeneration of the petrous bone and the labyrinth, which gave rise to a cerebellar abscess.

CEREBELLAR ABSCESS TREATMENT; IMPROVEMENTS IN TECHNIC WITH THROMBOSIS OF INFERIOR PETROSAL SINUS

Atkins [65] reported the case of a boy, aged 17, with acute middle ear suppuration. Antrotomy was performed about two weeks after the onset. There was initial improvement with abducens paresis. The internal jugular vein was ligated and

63. Calamida, U.: Some Rare Bacteriological Findings and Complications in Otogenic Thrombophlebitis, Orvosi hetil. **72**:600, 1928.

64. Rockemer, K., and Wirth, E.: Bacteriological and Histological Examination of a Case of Aural Actinomycosis with Cerebellar Abscess, Beitr. z. Anat. Physiol., Path. u. Therap. d. Ohres. **27**:10, 1928.

65. Atkins, R. Travis: Suppuration of the Petrous Pyramid; Rupture of Extradural Abscess Over the Apex: Suppurative Cerebrospinal Meningitis, Death, Autopsy, Laryngoscope **38**:404, 1928.

an incision made into the sinus. There was no visible thrombus. Symptoms of sinus phlebitis were noted. At a third operation, the dura was exposed over the middle and posterior cranial fossa. No pathologic process was revealed. Autopsy showed osteomyelitis of the petrous bone, extradural abscess, suppurative leptomeningitis and thrombophlebitis of the inferior petrosal sinus.

ABDUCTOR PARALYSIS IN RELATION TO GRADENIGO'S SYNDROME

Rudakov [66] said Gradenigo's syndrome of abducens paralysis and trigeminal neuralgic pains on the same side as middle ear suppuration has not a definite pathologic entity. The pain is due to a secondary meningitis, from ostitis of the apex of the petrous bone. From seven personal cases, he was unable to accept Gradenigo's explanations but thought that the abducens paralysis may have different causes; viz., may be of reflex origin, infectious-toxic origin due to a process in the temporal bone from an abscess.

CEREBELLAR ABSCESS: TREATMENT IMPROVEMENTS IN TECHNIC

J. V. Bissett of Newark, N. J., described a new device for controlling hemorrhage from the lateral sinus in operations for cerebellar abscess by pressure from two balls which are sewed into the dura. When both balls are in position, an opening into the sinus wall can be made between them, thus allowing a large incision of the dura.

Sojo [67] advocated opening through the mastoid process. The opening should be about two-thirds centimeter in diameter.

RESULTS OF TREATMENT IN BRAIN ABSCESS AND MENINGITIS

Among the otogenic brain complications in the Heidelberg Clinic during the years 1920-1926,[68] sinus thrombosis is recorded thirty-six times; twelve of the patients recovered. All the twenty-two patients with purulent otogenic meningitis died. Of five patients with cerebral abscesses, one recovered; all ten patients with cerebellar abscess died.

From 1900 to 1925, thirteen brain abscesses were opened in St. Joseph's Hospital, Copenhagen, and from 1912 to 1925, nineteen in the Rigshospital.[69] Of these thirty-two cases, the abscess was in the

66. Rudakov, V.: Otogenic Abducens Paralysis, Russk. oto-laring. **21**:62, 1928.

67. De Sojo, F.: Comments on the Treatment of Otogenic Cerebral Abscesses: Practical Remarks Based on Personal Experience, Rassegna internaz. di clin. e terap. **8**:685, 1928.

68. Behlau, C.: Statistical Data on the Otogenic Brain Complications Observed in the Heidelberg Clinic in the Years 1920-1926, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:26, 1928.

69. Norsk, F.: The Clinic of Otogenous Cerebral Abscess Illustrated by Cases at the Rigshospital and St. Joseph's Hospital, Copenhagen, Internat. Clin. **38**:166, 1928.

cerebrum in twenty-one and in the cerebellum in eleven. The lumbar fluid pressure was moderately increased as a rule; it was normal in only one instance; once it reached 1,200 mm. In three of sixteen patients, the fluid showed bacteria. The cellular content was greatly increased in seventeen. In seventeen of the thirty-two cases the fundus oculi was normal. The mortality was 75 per cent.

In St. Bartholomew's Hospital, eighty-five brain abscesses were observed in the last twenty-four years, with seventeen recoveries.[70]

VIII. LABORATORY AIDS IN DIAGNOSIS OF AURAL INTRACRANIAL COMPLICATIONS

BLOOD PICTURE IN AURAL SUPPURATION COMPLICATIONS

Kaiser[71] compiled eighteen cases from the University Clinic in Königsberg. He found that grave complications, such as meningitis, brain abscess, sinus thrombosis and extradural abscess always manifest themselves in some form in the blood picture, either by a greatly increased leukocyte count, a high neutrophilia or a left-sided displacement.

Kaiser attached great importance to the blood picture in determining whether the suppuration has been controlled after operation. Here it often is a better index of latent abscesses and extension of thrombus than are the fever and the pulse.

In intracranial complications the displacement to the left is the rule. This, however, is not invariable. The number of eosinophils is of special value.

A normal or slightly increased leukocyte count (below 10,000) in conjunction with a marked displacement to the left (around or above 20 per cent) permits in a general way the diagnosis of sinus thrombosis.

HYDROGEN ION IN THE CEREBRAL FLUID IN OTITIC COMPLICATIONS

Donadei and Sillingardi[72] discussed the value of the p_H, (hydrogen ion concentration), in the cerebrospinal fluid in otitis with complications. In nineteen cases of otitis media with intracranial complications the p_H of the cerebrospinal fluid showed a displacement toward the acid side in all except two cases. This displacement was most marked in leptomeningitis, less so in brain abscess and still less marked in septic thrombosis. The authors credit this procedure with diagnostic value.

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70. Scott, Sidney, in discussion on Sargent, Percy: Drainage of Brain Abscess, Brit. M. J. **2**:308, 1928.

71. Kaiser, P.: Examinations of the Blood Picture in Ear Complications, Arch. f. Ohrenh. **119**:237, 1928.

72. Donadei, G., and Sillingardi, G.: The Value of the p_H in the Cerebrospinal Fluid in Otitis with Complications, Arch. ital. di otol. **39**:216, 1928.

CHARACTER OF CEREBROSPINAL FLUID IN SUPPURATION

Buccelli [73] compiled and tabulated twenty cases of acute and chronic otitis in cerebral complications from the mildest form of meningismus to suppurative leptomeningitis. On the basis of the cerebrospinal fluid changes, two general groups can be distinguished: 1. In one group are the acute inflammations associated with a rise of fluid pressure, slightly increased albumin content, nearly normal glycogen content, negative globulin reaction and scanty lymphocytes. These usually have a favorable course. 2. In the other group are chronic inflammations (cholesteatomas) with intracranial complications, associated with rise of pressure, increase of the total albumin, diminution of dextrose, negative globulin reaction and scanty polynucleosis. Here the prognosis is unfavorable.

DIFFERENTIAL DIAGNOSIS BY THE CEREBROSPINAL FLUID OF ENCEPHALITIS FROM TUBERCULOUS MENINGITIS

The meningital manifestations from the ear are not infrequently confused with tuberculous meningitis, which also may be mistaken for brain abscesses. Retrouvey and Derville [74] reported two cases: One patient suffered from tuberculous meningitis, which was interpreted as a brain abscess; the other patient had a brain abscess, but was thought to have tuberculous meningitis. Stewart [75] said that in a case of doubtful tuberculous meningitis the cerebrospinal fluid should be examined for (1) protein, by qualitative and quantitative methods, and (2) the differential cell count.

The meningism of pneumonia or intestinal intoxication may increase the protein content of the fluid. In such cases, however, the cell count is not increased. In children the normal total protein in the lumbar region varies from 15 to 55 mg. per hundred cubic centimeters and in the cisterna magna from 12 to 50 mg. per one hundred cubic centimeters. Even in early cases of tuberculous meningitis it is raised above normal limits. In a series of forty-two cases of tuberculous meningitis, both lumbar and cistern fluids were examined bacteriologically.

Tubercle bacilli were found in the cistern fluid in 100 per cent, and in the lumbar fluid in only 57 per cent of cases. Tubercle bacilli were found in the lumbar fluids within forty-eight hours of admission in twenty-four among forty-two cases, that is, in 57 per cent. At the first puncture, bacilli were found in ten cases; at the second, in fourteen more.

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73. Buccelli, A.: Behavior of the Cerebrospinal Fluid in Aural Affections, Arch. ital. di otol. **39**:525, 1928.

74. Retrouvey, H., and Derville: Tuberculous Meningitis and Encephalitic Abscesses, Rev. de laryng. **49**:251, 1928.

75. Stewart, Donald: Cerebrospinal Fluid; The Diagnosis of Tuberculous Meningitis, Edinburgh M. J. **35**:141 (March) 1928.

However, in the cistern fluids the tubercle bacilli were found in every case within forty-eight hours after admission, that is, in 100 per cent. At the first puncture, organisms were found in thirty-four of the forty-two cases, in eighteen after centrifugating for from five to twenty minutes and in the remaining sixteen after incubating the fluids for twenty-four hours. At a second puncture, the tubercle bacilli were found in the other eight cases, after incubating the fluids for twenty-four hours.

DIAGNOSIS OF MENINGITIS FROM ENCEPHALITIS BY SODIUM FLUORESCEIN IN THE FLUID

Schippers and Peters [76] found that in the presence of meningitis, uranin (fluorescein sodium) given by the mouth, in doses of 20 mg. per kilogram of body weight, passes into the cerebrospinal fluid in concentrations greater than 0.04×10^{-5}. Lesser concentrations are not considered as pathologic. In some cases, a slight fluoroscence is noted with so-called "intact meninges." Fluorescein can be employed for the differential diagnosis of meningitis from acute encephalitis and a number of other diseases of the central nervous system. It is a valuable adjuvant in the diagnosis of meningitis.

Although the cerebrospinal fluid may be slightly fluorescent in a given case, the boundary value can be established and concentrations 0.04×10^{-5} are to be considered as nonpathologic; consequently the authors could establish 0.04×10^{-5} as the boundary value. It is necessary that the cerebrospinal fluid be perfectly clear and that the fluorescence is estimated in the dark chamber, with lateral illumination. In doubtful cases, it is always best to perform a caloric measurement. Even a small admixture of blood renders the determination too high, in which case another test may be done at the end of seven days, not earlier, because in pathologic cases, all the uranin has disappeared only at the end of five or six days.

IX. MENINGITIS OF OTITIC AND NASAL ORIGIN

TYPES AND FORMS OF MENINGITIS

Incidence of Different Types.—Neal [77] found pneumococcus the most frequent cause of suppurative meningitis. In ninety-eight cases the following types were found, type I, thirty; type II, seventeen; type III, twenty, and type IV, thirty-one.

In 123 cases of streptococcus meningitis, all but one hemolyzed with a proper technic, Neal emphasized the lack of meningeal symptoms in infants (no Kernig sign or stiff neck), and the prominence of gastrointestinal disturbances without vomiting.

Protective, Martland Serous or "Sympathetic" Meningitis.—A labyrinthogenic meningitis associated with deafness and caloric non-

76. Schippers, J. C., and Peters, H. J.: Uranin as an Adjuvant in the Diagnosis of Meningitis (Fluorescein Sodium); Nederl. Tijdschr. v. Geneesk. **72**:4514, 1928.

77. Neal, Josephine B.: Unusual Infections of the Central Nervous System, Arch. Neurol. & Psychiat. **21**:451 (Feb.) 1929.

irritability on the side of the healthy ear followed scarlet fever. Neuritis of the eighth nerve, as a sequel of the suppurative meningitis, was confirmed by autopsy and microscopic examinations. Guettich [78] proposed the designation of "sympathetic meningitis" for this type of lesion because of its analogy to sympathetic ophthalmia. Both parts of the eighth nerve on the side of the healthy ear may be affected, but more frequently only the vestibular nerve is attacked.

Moscardi [79] interpreted serous meningitis as caused by a few germs, penetrating by a blood or lymph route from the ear. They thus exert a toxic effect on the choroid plexus and ventricular ependyma and cause an increased production of cerebrospinal fluid with rise of pressure; hence the symptoms of severe headache, vomiting and vertigo. He stated that the symptoms of serous meningitis are: changes of the retina, papilla or macula, although vision is not affected. Occasionally there are paralyses of the external rectus. One of his cases also showed complete oculomotor paralysis. The fundus changes are of diagnostic and prognostic importance, in that those that point to continued pressure are the least amenable to treatment. The changes of the ocular fundus correspond to the side of the ear disease.

Operations on the ear and lumbar puncture modify the ophthalmoscopic picture. Thirteen of the seventeen cases presented retinopapillary edema of mild degree. In six of seventeen reported cases, a mastoid operation alone sufficed to cause all the symptoms, meningeal and ophthalmoscopic, to disappear. In three cases a cure which had not followed on the ear operation, was obtained by repeated lumbar punctures. Some cases were favorably influenced by lumbar puncture alone without operation.

"Interval" or Latent Meningitis.—Hazama [80] reported two cases of delayed meningitis. He would designate this type as "interval," because considerable time intervenes between the onset of the meningitis and the middle ear inflammation, during which the patients are entirely free from symptoms. The interval type is especially common with *Streptococcus mucosus*, but other germs have been found. The cause of the sudden onset of meningitis in one of his cases was a rupture into the labyrinth, and in the other rupture into the posterior fossa. Elderly patients are especially susceptible. Both of his cases followed acute middle ear inflammations in elderly persons (women 60 and 50 years of age). Microscopic examination showed that the process in the tympanum had healed, the ear was dry, but nevertheless there

78. Guettich: Pathological Anatomy of Sympathetic Otitis, Beitr. z. Anat. Physiol. Path. u. Therap. d. Ohres. **27**:6, 1928.

79. Moscardi, P.: Serous Meningitis of Otogenic Origin, Riv. oto-neuro-oft. **5**:293, 1928.

80. Hazama, T.: Origin of Late Meningitis in Acute Otitis Media, Monatschr. f. Ohrenh. **62**:680, 1928.

was an abscess in the bone at the posterior surface. In one case, *Streptococcus mucosus* was present in the lumbar puncture fluid; in the other case *Diplococcus lanceolatus* was found.

A woman had recovered from scarlet fever four years before she was seen by Batier.[81] A right-sided purulent otitis media developed four weeks before examination. Paracentesis was followed by a discharge which ceased after ten days. At the time of the examination, the tympanic membrane was bright red and bulged slightly; there was no discharge; the mastoid process was normal. Four weeks later, there suddenly appeared marked restlessness, vertigo and vomiting, followed by loss of consciousness and a positive Kernig sign. At operation, the mastoid process contained no pus, but granulations were seen on the floor of the middle fossa. Two days after the operation, the patient regained consciousness, but presented speech disturbances. She had difficulty in finding words although word recognition was prompt. The author expressed the belief that the latent mastoiditis must be referred to the scarlet fever four years before to explain the changes in the bone.

BACILLUS MUCOSUS CAPSULATUS (ALLIED TO BACILLUS COLI-COMMUNIS) IN THE CEREBROSPINAL FLUID

In Brain's and Valentine's case[82] organisms from both the cerebrospinal fluid and in the blood were found by culture, which resembled *Bacillus coli-communis*. Guinea-pigs inoculated in the peritoneum died after three days from general peritonitis. They contained the bacilli. The bacillus appears to be closely allied to Friedländer's pneumococcus, which belongs to a group the members of which show very varying degrees of pathogenicity. The authors stated that the agent is to be designated as *Bacillus mucosus-capsulatus*, and that no such bacilli have hitherto been found either in the blood or in the cerebrospinal fluid.

The patient was attacked in November, 1923, by right-sided middle ear suppuration; he was cured by antrotomy. In July, 1927, operation was again followed by recovery. In September, 1927, he was readmitted with distinct signs of meningitis. The cerebrospinal fluid was turbid and under a high pressure. In addition, there was a mucopurulent secretion from the nose; death occurred after a few days.

DIAGNOSIS OF MENINGITIS BY FLUID: ATLOIDO-OCCIPITAL PUNCTURE

Jacod[83] advocated atloido-occipital puncture as devoid of danger. Thirty-six combined punctures showed equal pressure in both lumbar

81. Batier, G.: Latent Mastoiditis-Meningitis, Operation, Recovery, Arch. internat. de laryngol. **7**:329, 1928.

82. Brain, W. Russell, and Valentine, F. C. O.: A Case of Infection of the Meninges and Blood Stream by the Bacillus Mucosus Capsulatus, Lancet **1**:855, 1923.

83. Jacod, M.: Means of Investigation Furnished by Atloido-Occipital Puncture in Otogenic Suppurative Meningitis, Especially in the Focal Form, Acta otolaryng. **12**:244, 1928.

and atloido-occipital fluid. On compression of the jugular vein, the pressure rose more rapidly in the fluid from the occiput. The chemical reactions showed no essential difference, but the lymphocytosis was more pronounced in atloido-occipital region. Furthermore, the quantity and quality of the leukocytes was much more changed in the fluid from the occiput because of its greater proximity to the inflammatory focus. The depth at the atloid sac is about from 1 cm. in children to 3.5 or 4.5 cm. in women and from 4.5 to 6.5 cm. in men.

In the course of puncture of the cisterna cerebellomedullaris in a case of temporal lobe tumor, bright red blood spurted in a jet under a high pressure (Steindl [84]). The patient died four days later. Autopsy showed that the left vertebral artery had been struck by the puncture needle; the medulla oblongata was displaced downward and toward the right. There was almost complete absence of the cisterna.

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84. Steindl, H.: A Case of Fatal Outcome of a Puncture of the Cisterna Cerebello-Medullaris, Deutsche Ztschr. f. Chir. **209**:97, 1928.

Abstracts from Current Literature

Ear

ACUTE MASTOIDITIS: PROBLEM OF "EARLY" OPERATION. P. HOPSTEIN, Monatschr. f. Ohrenh. **63**:30 (Jan.) 1929.

Of 686 patients with acute otitis media who came under his observation at the St. Maria-Viktoria Clinic in Berlin, Hopstein reports that 383 were healed by conservative treatment. Most of the patients had been under treatment before entering the hospital. The 303 patients operated on were in many cases in a serious condition necessitating immediate intervention. In fourteen cases the time of onset of the symptoms could not be traced, these were excluded from the statistics. In 82 of the remaining 289 patients more or less severe complications were present. Sixteen patients died; however in one case death was due to an intercurrent infection with scarlet fever and in another to tuberculous meningitis. The mortality for otogenous disease in acute mastoiditis was therefore 18 per cent, as compared to 28 per cent in cases with complications. For the purpose of discussing the question of "early" or "late" operation, Hopstein grouped his material in the following manner: In the first group of twenty-three patients, operation was performed during the first week of the disease. In six instances perforation had occurred. The mortality was 4.3 per cent as compared to 80 per cent in complicated cases. The following group of eighty-seven patients contained fourteen patients with perforation. In all these antrotomy was done during the second or third week of the disease. The mortality was 5.7 per cent, as compared to 18 per cent of patients with complications. Operation after the third week of the disease was performed on 179 patients; in sixty-one of these perforation had occurred; fifty-nine had complications. The mortality was 4.5 per cent as compared to 33 per cent in cases with complications. On the basis of his experience Hopstein considers the term "early" operation should be set aside. Operation in clinically mild cases solely on the basis of pain in the mastoid process and an increased temperature with otherwise normal condition is not justified. However, if the condition of the ear or general state of the patient warrants intervention one should not hesitate to intervene by antrotomy.

THE EAR LABYRINTH OF THE SHARK (LAEMARGUS BOREALIS SCORESBY, SOMNIOSUS MICROCEPHALUS). C. F. WERNER, Monatschr. f. Ohrenh. **63**:195 (Feb.) 1929.

Werner studied these ear labyrinths, which he obtained from the collection of ear labyrinths of various fish, preserved in the National Historical Museum of Goteborg. On the basis of his study he contends, that, although his specimens conform in most points to the description of such ear labyrinths given in 1905 by Stewart, the fact that the utriculi in the latter's description are separated, points to the possibility of Stewart's specimens originating from another fish, belonging to the Notaindes. Kolmer and Eisinger's specimens had been preserved in ice for months and were in a bad condition; nevertheless their report of the unusual dimensions conforms on the whole to the description of Werner. He further stresses the importance of using fish of approximately the same size and of taking their ontogeny into consideration, as the site of the brain is variable and the labyrinth does not grow in the same proportion as the rest of the body.

OTITIS AS COMPLICATION OF PNEUMONIA IN CHILDREN. L. HOFMANN, Monatschr. f. Ohrenh. **63**:237 (March) 1929.

On the basis of his experience, Hofmann states that acute otitis media is a frequent symptom accompanying pneumonia in children. The proportion was 3 to 1. Distribution according to age was irregular; however, he found most cases in children under 4 years of age. There was no fundamental difference in the

course of otitis in lobar pneumonia or bronchopneumonia, although it was seen more frequently as a complication in the latter disease. Of 179 cases, fifty-eight children developed otitis media, whereas in 275 cases of lobar pneumonia disorders of the ear were evident in only forty-four. Otitis media acuta simplex (or acute otitis media catarrhalis) was observed in fifty-two cases (or 19 per cent of all cases of otitis). Purulent acute otitis media was observed in 243 cases (or 81 per cent). Its occurrence was seen only in twelve cases as precursor of pneumonia, and it usually ceased rapidly. Likewise rarely was this disease seen on outbreak of pneumonia; only eighteen cases were reported; however it frequently persisted for a long time. As complication of pneumonia 191 cases demonstrated the frequent incidence. Its course was the most favorable. Indication for surgical intervention is rarely found. Otitis in this case may be classed among the symptoms of "second sickness" occurring after pneumonia, such as sore throat, pharyngitis, lymphadenitis and bronchitis, and expectant treatment is indicated.

METASTATIC OPHTHALMIA OCCURRING AFTER OTOGENOUS SINUS THROMBOSIS. I. SOMMER, Monatschr. f. Ohrenh. **63**:419 (April) 1929.

Sommer reports this occurrence in two adult patients, one of whom recovered, although with the loss of one eye. In both patients, the condition became manifest after acute otitis. In one other case, that of a child, aged 3½, purulent chronic mastoiditis after scarlet fever was evident. Several days after the discharge of pus, the left eye became involved and finally atrophied. Although microscopic or bacteriologic examination was not possible in this last case, Sommer considers it characteristic enough to be classed with the aforementioned cases. On the basis of this study and a review of the literature, Sommer says that during purulent mastoiditis, metastatic reactions in the eye bulb, retina and orbital tissue can occur, due to thrombophlebitis. As far as can be ascertained, acute mastoiditis is by far the most frequent cause. Necropsies have revealed that thrombosis of the petrosal sinus, either alone or together with thrombosis of other sinus, constitutes an especially favorable condition for metastasis. It most often occurs in the eye of the involved side, though the opposite eye may occasionally be stricken; rarely are both eyes involved. Persons of any age are affected. According to Sommer, an abscess can be found in the conjunctiva, the cornea or in the iris. In the back part of the eye, purulent chorioditis, metastatic retinitis and septic emboly can occur. In all these instances abscesses are formed in the vitreous body or the orbit. The prognosis, however, is not unfavorable and most patients recover.

REACTION OF LEUKOCYTES IN MASTOIDITIS. S. P. JACHONTOW, Monatschr. f. Ohrenh. **63**:503 (May) 1929.

On the basis of a review of the literature and his personal experience in seventy patients (in fifty of whom differential counts were made) Jachontow says that there is a relation between diseases of the ear complicated by mastoiditis and the morphologic aspect of the leukocytes. Neutrophilia or hyperleukocytosis as compensation of this condition can occur. Deviation of the neutrophil blood count toward the left is supported by the fragility test of the erythrocytes. During convalescence the leukocyte count is restored to normal. According to Jachontow this deviation of the neutrophils is a more or less reliable guide in diagnosis, if correlated with clinical symptoms. In the diagnosis of mastoiditis without purulent infiltration of the surrounding tissue or complication by intercranial processes, leukocytosis is of no especial importance. In general, increase of the percentage of eosinophils in the blood is of favorable prognostic value. Eosinophilia in scarlet fever recedes when mastoiditis becomes manifest. A septic blood condition in otogenous thrombophlebitis is an indication against operative intervention. Jachontow points to the necessity of repeated blood counts for the purpose of constructing a curve. He demands that these examinations should be added to the routine work at the otiatric clinic. The slightest suspicion of involvement of the hematopoietic apparatus in patients with ear trouble should warrant intensified care regarding examinations of the blood.

ABSCESS OF THE CEREBELLUM. A. REJTÖ, Monatschr. f. Ohrenh. **63**:670 (June) 1929.

Rejtö reports that within the last two and one-half years, operation was performed at his clinic in 158 acute and 243 chronic cases of purulent mastoiditis. Eighteen of these were complicated by an extradural abscess, twenty-two by sinus thrombosis, four by an abscess of the cerebrum and six by an abscess of the cerebellum. Of these six patients only one recovered. Rejtö considers Lemaitre's technic of operation the best. He especially stresses the importance of early surgical intervention before the onset of symptoms of meningitis.

MATHEMATICAL DETERMINATION OF ENTRANCE TO MASTOID ANTRUM. D. N. MATWEJEW and N. M. PAUTKIN, Monatschr. f. Ohrenh. **63**:730 (July) 1929.

Matwejew and Pautkin developed an apparatus founded on the measurements of fifty skulls, by means of which they were able to reach the antrum without difficulty, even when unusual bone formation was present. They also made forty operations with assistance of this apparatus. In every instance, also in patients possessing abnormal site of the antrum, they could reach the opening without injury to the tegmen tympani, nervus facialis, semicircular canal or the sigmoid sinus. Their mode of calculation is the following: The outer side of the temporal bone is seen to form, what the authors name, the temporal triangle. The points are given by the zygomatic process, the linea temporalis and the third, at the projecting point of the mastoid process. The mastoid antrum is now found in the direction of a perpendicular line, erected at the point of intersection of having of the temporal triangle. In Matwejew and Pautkin's specimens, this normal line was invariably next to the spina supra meatum Henle, and it led directly into the antrum. They contend that the geometric determination of the site of the antrum is effective, and that ample demonstration has been given of its usefulness before antrotomy to warrant this statement.

AURAL NYSTAGMUS: CASE. M. RUTENBURG, Monatschr. f. Ohrenh. **63**:746 (July) 1929.

Rutenburg's patient, a man, aged 30, presented symptoms of subacute purulent otitis media and mastoiditis of the same ear, coincident with dizziness, vehement vestibular disorder and nystagmus. Caloric test was positive for a fistula, although with a paradox result. Antrotomy disclosed, besides an extensive destruction of the temporal bone, a large fistula in the upper part of the lateral semicircular canal, which was covered with granulation tissue. According to Rutenberg, the paradox caloric test may be explained by a change of pressure in the labyrinth due to contraction of the blood vessels and shrinkage of the granulation tissue caused by the cold water. Subsequent reaction of the eyes toward the diseased side is evident, thus giving support to the theory of Mygind.

Pharynx

PALATINE TONSILS: DEPOSITION OF PIGMENT CONTAINING IRON AND PIGMENT WITHOUT IRON. H. BRUNNER, Monatschr. f. Ohrenh. **63**:141 (Feb.) 1929.

On the basis of a review of the literature and his personal study on twenty-five pains of tonsils derived from tonsillectomies and fourteen pains from necropsies, besides experiments on eleven rabbits, Brunner states that under normal conditions the tonsils have a low iron content. Iron is more often evident in material obtained from necropsies than in tonsils derived from tonsillectomy. It is accumulated on the borderline between lymphatic tissue and the capsule, in the lymphatic tissue itself and is also toward the outside of the follicle. Pigment without iron is found on the whole, in the same places, as pigment containing iron. Deposition of iron and pigment is most frequently due to resorption of a pathologic hemorrhage caused by trauma or an inflammation. It may also be associated to hemolysis occurring in the tonsils under certain conditions. Iron derived from

the blood circulation is likewise deposited. Following intravenous injection of a small amount of colloidal iron into rabbits, the iron is first deposited in the wall of tonsillar veins, then also on the wall of the capillaries. In this respect they are to be distinguished from the lymph glands. However, as the endothelium of vessels in the tonsils is impermeable for colloidal iron and accumulation of iron in the reticular cells is thus prevented, the tonsils resemble lymph glands, but are at variance with the behavior of the liver, spleen and bone marrow.

THROMBOSIS OF VEINS IN PERITONSILLAR ABSCESS AND SORE THROAT. R. WALDAPFEL, Monatschr. f. Ohrenh. **63**:295 (March) 1929.

Waldapfel presents microscopic evidence in support of his contention that the importance of thrombosis of veins has been greatly overemphasized. Its occurrence is frequent, he states, and may be found to a certain extent in any type of abscess. Examination in serial section was made of the tonsils of patients who had been suffering from peritonsillar abscess and in several cases, from lacunar tonsillitis. Waldapfel stresses the point that in spite of thrombosis and suppurative exudation into the vessels, all the patients have recovered and are now in good health.

FIBROMATOUS POLYP OF BASE OF SKULL: VALUE OF PRELIMINARY OPERATIONS. A. M. NIKOLSKY, Monatschr. f. Ohrenh. **63**:301 (March) 1929.

On the basis of his personal experience in eighteen cases and a review of the literature, Nikolsky advises to discard preliminary operation as often as possible and to resect the polyp by the natural way of entrance. In six instances he was forced to perform a preparatory operation. The type of this intervention varies according to each case. To prevent the occurrence of aspiration pneumonia, it is advisable to have the head in the position of Rose. Death occurs not so often due to loss of blood as to heart failure. The heart in these patients is greatly weakened due to the chronic oxygen deficiency incurred by their defective breathing. Chloroform anesthesia should be entirely discontinued and replaced by local anesthesia. In case of diffuse bleeding taking place after resection, he advises autotampon with the resected polyp. Ligation of the external carotid artery does not influence the flow of blood occurring during the major resection of the polyp and should be avoided, as it constitutes an unnecessary trauma.

SORE THROAT: CLASSIFICATION FROM POINT OF VIEW OF PATHOLOGIC ANATOMY. A. G. FETISSOW, Monatschr. f. Ohrenh. **63**:396 (April) 1929.

Fetissow defines as sore throat (angina) any acute, nonspecific inflammation of the entire pharynx or of certain parts of it such as the tonsils. He also discusses the terminology of the various symptoms. On the basis of a brief review of the literature and his personal study of four cases he contends that, theoretically, the most rational division would be into two groups. In simple or superficial catarrhal sore throat, an increased output of lymphocytes and leukocytes in the epithelium is seen, without causing, however, destruction of the epithelium; on the surface of the tonsils an inflammatory exudate is formed. Their tissue is swollen and edematous. In parenchymal or lacunar tonsillitis, destruction of the epithelium is effected. Ulceration has occurred and the entire tissue of the tonsil is involved by destructive suppuration. In this group the old terminology conforms with the microscopic picture.

RETROPHARYNGEAL ABSCESS AS A COMPLICATION OF MASTOIDITIS. J. MISSIONŽNIK, Monatschr. f. Ohrenh. **63**:481 (May) 1929.

Missionžnik recognizes five paths of entry for the spreading of pus from the apex of the mastoid process into the pharynx. He supports the theory of Mygind who sees in the incidence of a retropharyngeal abscess as complication of a peribulbar abscess, persistence of the symptoms characteristic of a carotid

abscess. Thus, high temperature, painful swelling of the carotid nerve and early differentiation of tissue occur. Abscess of the digastricostylopharyngeal muscle shows fewer external symptoms, but reactions of the posterior pharyngeal wall are evident. The most frequent type of abscess is the one found in the sternocleidomastoid muscle; it is characterized by high temperature, symptoms of torticollis, and circumscribed swelling of the retromandibular region and the carotid nerve. Reactions from the abducens and trigeminal nerve are evident in discharge of pus from the apex of the petrosal sinus. Lastly, a retropharyngeal abscess can be caused by suppuration of the retropharyngeal lymph nodes which are partly collectors of the lymph from the middle ear.

Larynx

NEURALGIA OF THE SUPERIOR LARYNGEAL NERVE. F. HUTTER, Monatschr. f. Ohrenh. **63**:402 (April) 1929.

On the basis of his experience in thirty patients Hutter says that pain on the outside of the throat is sometimes caused by "neuralgia" of the superior laryngeal nerve. Although genetic relationship between this sensation and diseases of the mucosa of the upper respiratory tract is not to be discredited, yet this pain is a separate manifestation for which the usual treatment is not efficacious. Especially typical are the histories of three patients in whom pain persisted for several months after tonsillectomy. Relief could be obtained only after specific treatment for neuralgia. In these cases the cause could not be a late reaction or radiation of pain from the operative trauma, but a disease of the nerve itself, although probably of a very mild type.

ORGANIC STRICTURE OF LOWER END OF TRACHEA AND BRONCHI: THERAPY. F. HASLINGER, Monatschr. f. Ohrenh. **63**:782 (July) 1929.

Haslinger discusses the respective treatment under various conditions and concludes his paper with, what appears to be, a complete list of the literature on each type of stenosis such as: congenital strictures, those due to compression by goiter, aneurysm; the thymus gland in relation to stenosis as well as diseases of the peritracheal and peribronchial lymph glands; mediastinal tumors causing stenosis; papilloma, sarcoma, carcinoma; strictures of the lower part of the trachea and the bronchi due to syphilis, scleroma or trauma.

Nose

OZENA: MODIFICATION OF METHOD OF TREATMENT ACCORDING TO HALLE AND LAUTENSCHLÄGER. M. J. CHARSCHAK, Monatschr. f. Ohrenh. **63**:318 (March) 1929.

Charschak describes a method of treatment which combines the Halle and the Lautenschläger operations for reducing the opening of the nose. After the usual preparation of the nose for several days previously, local anesthesia with procaine hydrochloride and epinephrine is administered. The face is laid open by an incision reaching to the bone, from the fold of the frenulum up to the second molar. The bone is now chiselled out, especial care being given to keep the mucous membrane of the mouth intact. This is now cut and the opening plugged with gauze to prevent the flow of saliva into the maxillary sinus. A vertical incision is made from the middle and inferior turbinal across the lower surface of the nasal duct to the septum. The lower surface and the wall of the duct are now subperiostally separated, about to the depth of the palatine bone. Through the lower part of the frontal opening, a chisel is passed up to the front part of the maxillary sinus. The mucosa is separated and the sidewall carefully pushed toward the septum. A conical roll of iodoform gauze is pushed through the nose, so that its end just touches the posterior wall of the maxillary sinus. Its lateral wall is thus pressed toward the septum. The mucous membrane is now sutured

from the mouth. The iodoform roll is left for nine days in the nose unless a reaction in the cheek occurs. If the reaction is slight, the iodoform roll is removed after the fifth day and replaced by iodoform tampons. On more marked reaction, the sutures are removed, but the roll is left in place until the ninth day. After its removal the lateral wall of the sinus is pressed toward the septum with a Killian speculum no. 2, and for two months iodoform tampons are placed every second or third day. Operation on the second side may be done two to three weeks after the first intervention. Charschak's experience covers in all twenty-two nasal cavities on thirteen patients. Three patients left the city and the results could not be traced; in the remainder the outcome was favorable.

PHARMCOLOGIC REACTIONS IN THE ERECTILE TISSUE AND SUBEPITHELIAL NET OF CAPILLARIES IN NOSE: VASOMOTOR NERVE SUPPLY OF THIS VASCULAR SYSTEM. H. STERNBERG, Monatschr. f. Ohrenh. **63**:390 (April) 1929.

Sternberg comments on the literature and concludes, on the basis of this study and the results of his experiments, that the erectile tissue and subepithelial capillary system of the nose often have a correlative action. Thus, contraction of the erectile tissue or of the mucosa may go hand in hand with contraction of the subepithelial vessels; paleness of the mucous tissue gives evidence of this fact. Likewise, dilatation of the erectile tissue or swelling of the mucous tissue is coincident with hyperemia of the subepithelial capillary system and is seen by intensive coloration of the mucous tissue. But these vascular systems may evince an independent state of blood supply and, as Sternberg's experiments have shown, may even present divergent reactions. In view of these facts, erectile tissue and the subepithelial net of capillaries, according to Sternberg, must be conceded to possess dissimilar areas of sensitivity and reactions for stimulants of nervous or pharmacologic origin.

ORIGIN AND SURGICAL CORRECTION OF DEVIATED OR LUXATED NASAL SEPTUM. J. WELEMINSKY, Monatschr. f. Ohrenh. **63**:530 (May) 1929.

Weleminsky supports Menzel's theory that the embryonic anlage is responsible for anomalies in a number of cases. The existing method of operation is not satisfactory because there is too little mucous membrane on the side of the luxation. The operative technic described by Weleminsky consists in retaining the cartilaginous support of the septum and thus avoiding the irritating double tampon. An incision is made on the side of the luxation, approximately parallel with the fundus of the nose and about 1 to 2 mm. below the upper border of the frontal part of the vomer and the spina nasalis anterior. It reaches through the mucous membrane and cartilage to the frontal part of the septum and is extended through the horizontal part of the mucous membrane. The cartilage is detached from the wall and reset on the bone on the inside, thus preventing the occurrence of a spontaneous dislocation. The mucous membrane on the opposite side is likewise detached from the vomer and the spina. It is important to bend the lower edge of the cartilage round because the ensuing arch in the lower part relieves the tension in the upper part. A shallow S shape of the septum is thus formed which eventually becomes compensated. The cartilage which has remained on the underlying part is resected submucously. The fold in the mucous membrane usually becomes equalized but it can be cut away and one or two sutures placed. There is no need for a tampon, a fact which Weleminsky ascribes to the treatment of the wound with trichloracetic acid. In a patient with evidence of a previous unsuccessful operation, Weleminsky suggests resection of the crooked part of the bony septum as the simplest method of treatment.

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

ROBERT SONNENSCHEIN, M.D., *Reporter*

Sixty-Second Annual Session Atlantic City, N. J., May 22, 23 and 24, 1929

J. GORDON WILSON, M.D., *President, in the Chair*

Wednesday Afternoon Session, May 22, 1929

OTOLOGY AND NEUROLOGY. DR. J. GORDON WILSON, Chicago.

This article will appear in full in a later issue.

EARLY STAGES IN THE DEVELOPMENT OF THE ACOUSTIC APPARATUS IN THE HUMAN EMBRYO. DR. GEORGE L. STREETER, Carnegie Laboratory of Embryology.

Of the various sense organs the ear is the first to make its appearance in the human embryo. As the first somites begin to show, and before the closure of the neural tube, that part of the neural wall that is to furnish the acousticofacial ganglion exhibits an active proliferation of its cells, producing a lateral bulging. From the lateral surface of this fold the ganglion mass becomes detached by delamination. Coincident with this, an area of thickened skin ectoderm becomes demarcated as the otic plate. The progressive differentiation of this otic plate and its invagination and detachment from the surface ectoderm have been photographed and demonstrated in a series of twenty-four embryos, ranging from the stage of 7 somites to embryos 4 mm. long, at which time the otic vesicle is completely established, and the walls are histologically differentiated into parts that can be clearly recognized as the vestibular, cochlear and appendicular portions.

After the completion of the primary vesicle and its ganglion, one can begin to see changes in the mesenchymal environment, at first a condensation of cells and soon a definite capsule of precartilaginous tissue. This capsule, from the first, assumes definite relations to the membranous labyrinth, and one finds the endolymphatic appendage not inclosed. The latter is left naked to come into relation with the lateral sinus. Along with the development of the precartilaginous tissue a reticular space, intervening between it and the epithelial labyrinth, makes its appearance and this reticulum can be traced into the eventual periotic spaces with their equipment of blood vessels. In the meantime, the precartilaginous tissue becomes definite cartilage.

The third developmental period concerns the transformation of the cartilaginous capsule into bone, which is covered by the investigation of Dr. E. H. Bast.

OSTEOGENESIS OF THE HUMAN PERIOTIC CAPSULE. THEODORE HIERONYMUS BAST, Department of Anatomy, University of Wisconsin.

This article was published in full in the November issue, p. 459.

DISCUSSION

DR. J. GORDON WILSON, Chicago: We have received one of the best descriptions of the development of the ear that has ever been given in this country. I have listened to the expositions by Dr. Streeter and Dr. Bast with great pleasure. The enthusiastic reception given these papers is evidence of our appreciation.

DR. STACY R. GUILD, Baltimore: I can only express admiration for Dr. Streeter's work. I have seen many embryos in animals but I have never seen such a complete story as he has given with human embryos. One point is that regarding the peculiar weblike bone described by Max Meyer. Dr. Bast says that this is primarily endochondrial bone, and I am inclined to agree with that. It impressed me as having the value Meyer attached to it. It looks rather like our old embryonic friend. I think also that there is an intrachondrial (not tangled with endochondrial) characteristic formation of the spaces. I should like to ask if you interpret the part near the cell as calcified matrix. The pictures look like what we get all the time. Why is that so different in amount in different persons, irrespective of age, whether new-born infants or adults? Even the amount of the interglobular spaces is different. Sometimes they predominate the picture; sometimes they occur only occasionally. As to the question of mitosis, I cannot say one way or the other, but it has some proportion of the growth in the ear, but I think that it is a secondary feature. In regard to the relation to otosclerosis, which is an abnormal development of some kind, I think that it is important to have this normal growth for comparison. I do not know whether we can find embryos in the early stages. Usually abnormal conditions lead to otosclerosis. The slides show Cozzolino's zone in otosclerosis. We have found histologically bilateral otosclerosis in an infant, aged 1 year and 10 days. In Cozzolino's area the fissura has been considered the focal point for development of otosclerosis. There is always great venous stasis over an area of otosclerosis, and Wittmaack considers that this venous stasis is associated with otosclerosis. In one infant, a slide showed otosclerotic areas at the superior part of the stapes and the anterior part of the round window. Under the superior margins of the stapes there also was vascular tissue. The anterior border of the round window was separate from the other foci. The condition was bilateral. The interglobular spaces were scarce.

DR. J. HOLINGER, Chicago: The earliest recorded case of otosclerosis was one noted by Alexander, in a child of 1 year, as told me by Siebenmann. The islands of cartilage between the bony capsule are remnants of the primordial capsule of the labyrinth. Siebenmann showed a number of these. Sclerotic foci which have not developed to the point of showing bone-marrow may be seen, and can be identified by the larger bone corpuscles. Bone corpuscles in the sclerotic bone are larger than those in the normal bone.

DR. G. E. SHAMBAUGH, Chicago: I find myself confused in regard to one point in the development of the osseous capsule: when a section is cut through the promontory three layers are seen: (1) cartilage, (2) periosteum lining the tympanum and (3) the lining of the perilymphoid spaces of the scala tympani. I did not hear these three layers mentioned, but perhaps I am mistaken.

DR. T. H. BAST: Dr. Shambaugh's question is easy to answer. All the cartilage except certain elastic fiber cartilages, is related to bone growth, the internal perichondrium next to the canals of the vestibule and cochlea and the external perichondrium which becomes internal and external periosteum. The external periosteum is more active in laying down bone. It is thicker on the outside and thinner on the inside of the endochondrial bone. Dr. Guild referred to the intrachondrial bone related to the calcified cartilage matrix. It is old cartilage matrix which has become calcified. This becomes much more voluminous than the actual cartilage was at the beginning and encroaches on the lacunae. I was asked why we do not get further excavation in other bones, but cannot answer this question. My personal opinion is that in case of otosclerosis a secondary process begins but there is a delay or checking in the growth. Why it does not go on normally I do not know, but it looks like a reexcavation and filling in of bone. I think that otosclerosis is a secondary excavation process.

DR. J. GORDON WILSON: Would that account for the cartilage at the foot plate of the stapes being ossified?

DR. T. H. BAST: That would be my impression.

Dr. George Streeter, Baltimore: Dr. Guild raised a point as to the relation of the mesenchyme and neuroderm to the periotic tissue. In some animals a large part of the cartilaginous cranium is of mesenchymal origin. It is natural to expect that this capsule might be of this type, but it is possible that the otic capsule is ectodermal in origin. In fishes with cartilaginous skulls the otic apparatus is the most antique primary sense organ, developing much before the eye, for instance. Perhaps the capsule may be related by invasion of other tissue.

Demonstration of Wax Plate Model of the "Utriculo-Endolymphatic Valve" (Bast) in a Two Year Old Child. Dr. J. Gordon Wilson, Chicago.

The fold of the posterior utricular wall, placed at the opening of utriculus into the endolymphatic duct, and described earlier by Bast as occurring in the human embryo, has been found as a striking structure in a series of sections prepared from the ear of a child aged 2 years.

In sections, the fold appears as a shank with a footlike free extremity; the latter is turned forward and, in our series, is applied to the utricular wall. The fold, at its greatest extent, is 0.717 mm. in length. As a free projection, either through the foot or both foot and shank, it occurs in thirteen serial sections, each 25 microns in thickness.

It consists of an epithelium, varying regionally between flattened cuboidal and low columnar, and an areolar tissue core, thin meshed except immediately beneath the epithelium of the shank and throughout the pedal portions, where it is concentrated.

The fold and adjacent parts of the membranous labyrinth have been modeled by the Born wax plate method.

DISCUSSION

Dr. Stacy R. Guild, Baltimore: I saw Dr. Anderson demonstrate this at the anatomic meeting. I have expressed my views in regard to its hydrostatic function. I cannot see how the perilymphatic fluid would pick it up. There is another point, namely, its similarity to that part in the guinea-pig model. All descriptions call for the duct from the saccule to join as a Y. All the classic accounts show a Y. This model shows a right angle junction as in the guinea-pig. I think that that is the usual form and that the other is due to poor technic. All the other models were made by taking bone off and conclusions were drawn from these fine membranes. You can perceive the difficulties of interpretation. Using good technic, as in this case, explains it. I should like to know how you get it, if you know.

Dr. J. Gordon Wilson: It was more by accident. We are now modeling this in a different way. We are modeling the structure of the endolymphatic system to show the dilatations and branches, and when we get it, we shall be able to show it.

Dr. H. T. Bast, Madison, Wis.: In regard to the hydrostatic function, I made it clear that this was a conjecture. Dr. Wilson has brought that out. I have no notion as to what the function might be. Regarding the Y shape, I have a number of preparations showing that the branch of the endolymphatic sac has one going to the utricle, one to the saccule. Some show a Y, others do not. I know that some of my technic is faulty, but I have obtained both. When I obtained a Y my histologic picture was better than in the other cases.

Dr. E. P. Fowler, New York: One point about the hydrostatic pressure may throw light on the subject. The sections might not show it acting as a valve, but as it communicates through a narrow opening with a large opening, you will get a section which will start a valve motion. Dr. Bast spoke of the shape of the duct as fan-shaped close to the thin membrane, but the thinness of the membrane would not prevent a valve action.

Dr. J. Gordon Wilson: The full description of the technic will be printed in the paper when published in the Transactions.

Thursday Morning Session, May 23, 1929

REPORT OF COMMITTEE ON THE STUDY OF PROGRESSIVE DEAFNESS. ARTHUR B. DUEL, New York.

On behalf of the Committee, the following report was presented:

The work of the Committee up to November, 1928, has been reported. The subsequent work has been a combination of all the projects we have had in hand. The report on otosclerosis has been completed, and the published volume is ready for distribution. We have received subscriptions from seventy-five members of this society and from the Triological Society. The work of translating, publishing and preparing the bibliography has cost $12,000, and the volume is being sold at $15. The monetary return is small, but we are seeking to increase interest in the subject. A review of all the literature on this subject will appear from year to year as a supplement to this volume. This may appear at three year intervals. We have also arranged with the Department at McGill University, under Dr. Birkett, Dr. McNally and Dr. Squeaver, to review the work done by Dr. Crockett on calcium in diet with reference to otosclerosis, together with a definite program of extension of that work along the line of dietary measures and parathyroid therapy. In this research a set of experimental animals are to be kept in a state of hypocalcemia and also in a state of hypercalcemia, in order to make histologic studies on the temporal bones in these conditions.

Besides the Research Council we have a Scientific Committee and a committee of lay members. An additional fund of $500,000 is to be collected for this research work. Mr. Harkness has promised help if this fund is collected by July 1. Dr. Dench has been active in this matter and has secured a promise of a residuary estate, worth not less than $50,000, which Mr. Harkness is willing to accept as if it were a pledge. This brings the total to $440,000. We have also approached the Carnegie Fund for help. They would allow us to use the unexpended sum on hand, plus the last contribution, $10,000, which would add $20,000 more. This leaves a deficit of $40,000 short of the goal. We have become ambitious and hope that we shall finally get $2,000,000, so that the work may be much prolonged and enlarged, and that its scope may include work in foreign countries with the prospect of getting much better and quicker results.

AN ENDOCRINE STUDY OF A THOUSAND CASE HISTORIES. DR. DANA DRURY, Boston.

Drury holds that subacute and chronic nonsuppurative disease of the middle ear falls correctly into two groups: catarrhal inflammation and otosclerosis. These show similar symptoms and may be coexistent. The secret of successful treatment for catarrhal disease lies in the early correction of abnormal conditions in the upper air passages.

There is a definite relation between disorders of function and organic defects. A large series of cases was studied from this standpoint. In more than 1,000 cases, 15 per cent showed thyroid disease, 80 per cent of these being hypofunctional. Of the thyroid group, 18 per cent gave deafness as a presenting symptom, 34 per cent gave a history of tinnitus and 15 per cent of vertigo. Two typical cases are cited, both showing improvement on thyroid medication. In the series of cases studied, 29 per cent showed pituitary involvement. Deafness was present in 22 per cent of these, the pituitary disorder being of the dysfunctional type. Thirty-six per cent gave a history of tinnitus and 24 per cent of vertigo. Two cases of otosclerosis showing improvement on pituitary medication are cited. In the same series, 12 per cent exhibited symptoms of ovarian hypofunction. Deafness was a presenting symptom in 13 per cent, while 34 per cent showed tinnitus and 22 per cent vertigo. The nonendocrine group (42 per cent) showed deafness in 19 per cent, tinnitus in 31 per cent and vertigo in 20 per cent. Deafness occurred more frequently in the cardiorenal group, tinnitus in the group of neuroses and psychoses and vertigo in that of lesions of the central nervous system.

Dr. Drury believes that when a correlation exists between deafness and a lowered basal metabolic rate, it seems most probable that both are results rather than causes. It seems fair to presume that when there is no demonstrable direct mechanical cause for the deafness the lowered hearing failure may arise from the lowered body metabolism due to the endocrine condition. Experience has shown this to be true with an appreciable percentage of otosclerotic patients.

DISCUSSION

DR. J. A. STUCKY, Lexington, Ky.: This paper shows a decided progress toward the solution of the problem of chronic deafness, confirming the clinical conclusions that I have held for many years. Experience has strengthened the opinion I advanced several years ago, that this condition is not due to a definite pathologic change but is primarily the result of deficient nutrition; toxemia, intestinal in origin; circulatory disturbance, and endocrine dysfunctions, which are the end-results of the deficient nutrition and toxemia. The hereditary tendency is probably due to prenatal deficiency.

In the majority of cases I meet there is clinical evidence of dysfunction of one or more of the endocrines, thyroid, gonad, suprarenal and pituitary, and most satisfactory results are obtained from pluroglandular feeding, but the toxemia must first be eliminated and the treatment supplemented by a balanced diet and biologic living, the latter being the most important part of the treatment.

Endocrine imbalance may be the result of hypofunction or hyperfunction and may involve either hearing or sight or both, and a careful checking up of the case at least once a week is necessary while administering the endocrine feeding, until the imbalance is corrected.

In each case of chronic deafness, I consider: (1) nutrition, (2) elimination (and constipation should be relieved without purgatives), (3) pulse rate and blood pressure and (4) a vasomotor system. My experience is that the majority of cases of chronic deafness can be arrested and can remain so as long as the patients live properly. The condition of many of the patients under 45 can be improved. The three most important endocrine glands involved are the thyroid, suprarenal and gonad. The graded endocrine feeding must be continued for some months until the proper functions of these glands are restored, and it must be remembered that a balanced diet must be continued in order to maintain this restored function.

DR. THOMAS J. HARRIS, New York: This paper deserves earnest commendation. I congratulate Dr. Drury on his careful study in regard to the endocrine system. We have not been so successful in our attempts. I am reminded of the paper read by Carlson before the society at the meeting at Swampscott. He said that with the exception of the thyroid gland we have not advanced far in the knowledge of the endocrine system. So far as Dr. Drury has spoken of the thyroid I am in agreement with him. I agree with Dr. Drury, Dr. Kopetsky and Dr. Bryant that the term catarrhal deafness of the middle ear should be discarded and that it is misleading. The term otosclerosis is itself a misnomer. We would get further in our study of chronic deafness if we omitted that term. Dr. Drury has brought out the close relationship of chronic deafness to the endocrine system, and has found, as we have, that the thyroid is most generally at fault, and more often with hypofunction than with hyperfunction.

I think that Dr. Drury is to be congratulated on his success in his studies from the therapeutic standpoint. In these cases we have not met much encouragement.

DR. L. W. DEAN, St. Louis: The second case that Dr. Drury presented showed a Ménière's symptom-complex. If one reviewed the literature of the subject for the last thirty years, one would find two other reported cases that were thought to be due to hypothyroidism. Dr. Drury reported these. Another one appeared in the English journals. I should be interested if Dr. Drury would elaborate in his closing discussion on the chemical studies he has made on the blood metabolism in general and its relationship to disturbance of aural function.

DR. S. MACCUEN SMITH, Philadelphia: A few years ago I reported a series of four cases in connection with Dr. Robert L. Bitfield of Philadelphia, in which the patients suffered from a tinnitus aurium and deafness, which were relieved by thyroid therapy; therefore, the conditions must have been due to dysfunction of this gland. Within the last few months, I have been associated with another physician in the care of a young woman suffering from a considerable loss of hearing and marked tinnitus aurium. She had been married for eight years but had never been pregnant and underwent an operation for the purpose of promoting conception. Following this operation she became pregnant, and instead of this condition producing an increase in her aural symptoms, as is so frequently the case, she has actually improved with reference to both tinnitus and hearing. In my experience the aural symptoms incident to myxedema are generally improved by thyroid therapy.

DR. E. P. FOWLER, New York: It seems rather lacking in appreciation even to suggest any adverse criticism of such a wonderfully careful and complete paper, but there is one point to criticize. It is a remarkable thing that in cases of chronic progressive deafness the great majority of people do not show any endocrine symptoms, or give an endocrine reaction. If they are examined carefully, their hearing may be found reduced, but the nerve of hearing is not usually affected, as evidenced by the fact that the bone conduction is normal. Perhaps myxedema affects only the capsule of the labyrinth or the conduction mechanism, but it is impossible to conceive how this can happen. Until we arrive at a point where we can find evidence of endocrine disturbance in the other parts of the body, it is stretching the point to say that those cases of ear trouble are of endocrine origin.

DR. H. H. VAIL, Cincinnati: I feel much as Dr. Fowler felt; namely, that if we accept all these facts we shall have to discard our armamentarium and become endocrinologists instead of otologists. I was impressed with the fact that much more attention was given to the laboratory examination than to the examination of the ear and sinus. I have tried similar treatment and I have given it up except in cases of obvious endocrine disturbance in which any one could make a diagnosis. I think that often there is a latent sinusitis back of the progressive deafness. All these patients give a history of frequent colds. Before one makes a diagnosis of endocrine disturbance as the cause of the deafness, I think that one should examine more carefully the parts around the ear. I have been associated with Dr. Cushing in cases that were obviously of endocrine disturbance, with gigantism, acromegaly or hypopituitarism, and have not seen any marked deafness. I have seen many cases of thyroid disturbance, but do not think that deafness is any more marked in these cases. I hope that Dr. Drury will not go too far in this endocrine therapy and neglect careful examination of the parts about the ear.

DR. C. C. BUNCH, Baltimore: I have sketched here an audiogram which shows the results of tests on a colored woman, aged 30. She had been in the hospital a few weeks previously for a lobectomy, the basal metabolic rate at that time being plus 51. When she recovered from the operation, she noted some loss of acuity of hearing. The audiogram showed rather marked bilateral loss which was fairly uniform for all tones. The drum membranes were normal, the Rinne test was questionably positive and bone conduction was not decreased. The results of the tests made us think that we might be dealing with a case of beginning otosclerosis. She was referred back to the thyroid clinic for a check of the basal metabolism. At this time it was minus 36. Under treatment prescribed by the thyroid clinic, her hearing began to improve and after some time became practically normal. The thyroid treatment was then discontinued. One month later, hearing tests showed that acuity for high tones was beginning to diminish. These results give rise to some interesting hypotheses.

DR. J. K. M. DICKIE, Ottawa, Canada: The subject of progressive deafness is extremely interesting. In my practice two of every three deaf patients are undoubtedly suffering from otosclerosis. There is a striking clinical aspect to

these cases. The patients appear to be decidedly below par, and give one an impression of diminished physical vigor. The voice is flat and toneless, and there are many indefinable symptoms. I think that in a large number of cases investigated by internists there is a general metabolic disturbance. In some cases there is a slight hypothyroidism. It appears to me from a study of otosclerosis that there is a hereditary degenerative tendency which may be excited by various causes—focal infection, endocrine disturbance, metabolic disturbance—but until now our various researches have not reached any marked result. We know what the histologic changes occurring in otosclerosis are, but there is no agreement as to what these changes mean.

Dr. L. W. Dean, St. Louis: I saw the case mentioned by Dr. Bunch, and have also had patients of my own with this condition. I have seen patients with a Ménière syndrome in whom examination by a competent internist was negative except for hypothyroidism. These patients improve so far as vertigo and loss of hearing are concerned after the administration of thyroid extract. My experience has been rather contrary to that of some of the other speakers.

Dr. Stacy R. Guild, Baltimore: We have prepared models that have a bearing on this question of return to function. We must, however, draw a line between clinical otosclerosis and true or biologic otosclerosis. I should like to report an interesting case of a patient with true otosclerosis with ankylosis of the foot plate of the stapes, who had no sign of clinical otosclerosis. The patient was a very sick woman (a cardiac nephritic patient) with otosclerosis on both sides, involving anterior and inferior to the stapes, to half the circumference. The anterior cruz was bound in with fibrous tissue in which there were deposits of calcified material independent of the stapes and of the opening of the oval window. The cartilage on the rim of the oval window was changed to sclerotic bone, and two areas showed partial ankylosis. This is a case of true otosclerosis just at the point of ankylosis, but with absolutely no indication clinically of an otosclerotic type of deafness. This is of interest in connection with otosclerosis with a lowered endocrine function of any type. I would raise the question whether true otosclerosis does respond to these changes, after bony union takes place. I cannot conceive of a return to a true annular ligament. Can a patient with such a case improve?

Dr. Dana W. Drury: There were no cases of hyperthyroidism in the group studied. Also, there were no cases of hypergonadism. In fact I have never seen such a patient complaining of ear symptoms. In a series of 3,000 case histories, there were only 18 cases pluriglandular in nature. The 18 cases were pluriglandular because the patients had been operated on, i. e., either the gonads or the thyroid had been removed before the endocrine study was made at the Evans Hospital. To evaluate the true metabolic rate means several determinations together with the pulse rate and temperature and respiration plus the person's weight and sitting height.

Classification of Chronic Deafness. Dr. James G. Dwyer, New York.

For years Dr. Dwyer has felt that the classification of deafness is not only an unsatisfactory one, but in the light of modern work on deafness it is positively misleading. He made a plea for a classification that will convey to the younger men entering the field of otology an idea of the pathologic process underlying deafness, so far as it is now known.

Take for instance the diagnosis of otitis media catarrhalis chronica. This classification includes numerous subgroups, and the term catarrhal is misleading. Again most modern otologists are agreed that many of the classes of cases fall under the focal infection type, and the usual term describing it not only is a misnomer but is misleading.

One could go through the whole classification and in the light of modern work could improve on it. Dr. Dwyer, therefore, made an appeal that deafness should be reclassified and the mental attitude readjusted to the whole subject.

DISCUSSION

DR. ARTHUR B. DUEL, New York: In the hospitals we have by routine practice used for diagnosis such terms as: otitis media catarrhalis chronica; otitis media catarrhalis acuta; otitis media purulenta chronica, and otitis media purulenta acute. Careful consideration of these cases makes it evident that such a nomenclature is incompetent for all cases. I believe that the term "catarrhal otitis media" should be removed from the nomenclature of diseases of the middle ear. They are hyperplastic, acute or chronic, or atrophic, acute or chronic. I doubt that a hyperplasia ever changes to an atrophy. As to the diagnosis of lesions of the eighth nerve I think that one should differentiate between the various types. In a class by themselves are those cases in which the degeneration of the nerve is due to toxic products brought from focal infection in other parts of the body. A neuritis due to a specific cause such as syphilis, lead or alcohol should be differentiated from the more common toxic cases originating from focal infections. I should like to see cases of otosclerosis carefully differentiated, and would postulate as one of the important diagnostic factors, a distinct hereditary history. If I were to make a change in the nomenclature, I should use the term otospongiosis instead of otosclerosis, as possibly conveying a better concept of the pathologic change. We might well appoint a commission of competent men to study a new nomenclature for diseases of the ear.

DR. L. W. DEAN, St. Louis: My classification of deafness, as it exists at present, without serious thought, is based on history and examination. As I look back on the way in which my patients are handled, I find that in later years a minimal time of twenty minutes has been given for taking every otologic history. I find that I must take that time in order to get a proper conception of the factors which bear on deafness. Unfortunately, it is difficult to get a familial history and data in regard to instances occurring early in life, even if they are extremely important. In such family histories we have found many endocrine disturbances. My classification is also based on a thorough general examination. Allergy is looked for among other things. To my mind, classification of deafness should be based on etiologic factors determined by the history, on general examination and on a thorough local examination which should provide the basis for therapeutic measures.

DR. FRANCIS P. EMERSON, Boston: I think that it is a distinct advance in the progress of otology when we acknowledge that our previous classification of the pathologic change of the middle ear has been unsatisfactory.

I have been much dissatisfied with the nomenclature of disease of the middle ear, because I have never been able to differentiate changes in the middle ear from those taking place in the perception apparatus. In one case we are dealing with physics, and in the other case with the nerve organ subject to injury, either of local or of general origin.

In syphilis, there is a specific, toxic injury to the eighth nerve. In mumps, we may get a dead nerve from one acute infection. In infectious disease there is toxic injury to the perception apparatus. There is dysfunction of the endocrine system in many diseases that may affect the eighth nerve. From my point of view I have worked out the following classification: Fundamentally, we have differed in our conception of disease of the middle ear from our difference in understanding the term catarrhal. This is a low grade infection, as has been shown by Zuckerhandel, Uffenordi and Hirsch. They have shown that the mucous membrane is markedly swollen to many times its normal thickness, so that it becomes almost jelly-like, and this is followed by an exudate or a serous effusion.

Regarding the classification of tubotympanic catarrh, we were taught that there was first a nasopharyngitis with involvement of the eustachian tube, especially at its mouth. The lining membrane became markedly swollen so that the tube became obstructed, and there was then a negative pressure and effusion into the middle ear. If we considered a catarrhal process and infection, we would

have a nasopharyngitis with involvement of the eustachian tube, which became markedly swollen followed by a serous effusion into the middle ear, without the necessity of conceiving that any negative pressure took place.

The next classification to which there is objection is that form of middle ear in which we have felt that there was a catarrhal, adhesive process. In this type of middle ear the drum membrane is clear. The middle ear is normal and there is no obstruction in the eustachian tube. We cannot conceive of a process involving the connective tissue without fibrosis and infiltration. This means increased blood supply, which again means exudation or secretion, which would involve the lining membrane. This we know does not take place; therefore, there cannot be such a pathologic process. On the other hand, we know that this classification covers a large number of cases. Then what is the etiology? If we go back to the primary classification of tubotympanic catarrh, we know that with the initial attack there is involvement of the middle ear and a marked lowering of tone perception. In a large number of cases this clears up, leaving no pathologic process in the middle ear and no damage to the perception apparatus.

On the other hand, there are certain cases in which the middle ear and the eustachian tube become normal and in which tone perception returns to normal for the low limits, but in which there is a distinct loss for the whispered voice. This loss progresses intermittently without disturbing the low limits or the middle ear. The cause is a bacteriemia or toxemia from the exacerbation of a local process, usually in the fauces, and we know this to be so, because with every acute exacerbation there is tenderness of the gland under the angle of the jaw. It is said that this is not nerve deafness, because there is no lowering of bone conduction, but there is never any lowering of bone conduction in any case from any cause until perception for the whispered voice is nearly lost, when the low limits begin to be raised, and when the patient no longer hears a tuning fork below 256 double vibrations. Then begins a lowered bone conduction in every case.

In other words, in every infection that involves the middle ear, whether it is a catarrhal process or a more virulent attack, there is at the same time damage to the perception apparatus, which is intermittent and in which the loss of tone perception takes places in an orderly fashion, until we finally get lowered bone conduction in all. The changes in the middle ear are coincident with the damage to the perception apparatus and cannot be separated from it.

Dr. E. P. Fowler, New York: There are two or three points that I wish to comment on. Of course everybody puts down otitis media catarrhalis chronica. It is a definite entity, and some diagnosis must be made. This designation simply means that it is not a suppurative case. I personally prefer the term otitis media nonsuppurativa as being most logical. We have only the main clinical and pathologic groups. No one term will cover every grouping. The best we can do is to get a nomenclature that will not mislead. In regard to the diagnosis of specific deafness from the audiograms, just because a patient has a positive Wassermann reaction does not mean that the deafness is caused by syphilis. Otosclerosis cannot be diagnosed from the audiogram alone. Adhesive lesions will give audiograms similar to those of otosclerosis. I have looked at thousands of slides of ossicular joints of animals with disease of the ear and have never seen any joint involvement, so that it would seem not to be common. The joints are apparently remarkably protected.

Dr. J. Holinger, Chicago: In both Dr. Dwyer's and Dr. Drury's papers there was no mention of comparative hearing tests. They would have given some idea about the function of the ears. The Rinne, Weber and Schwabach tests, and the lower and the upper tone limits are of greatest importance, especially if the different test changes. Middle ear deafness was spoken of, but the fact is that careful pathologic and statistical examinations have shown that the nervous system is more frequently affected than is suspected. Nerve deafness in all its different forms is not diagnosed often enough. Siebenmann has shown that nerve deafness

can be diagnosed by means of the tuning forks. These diagnoses were confirmed by pathologic specimens. The number of postmortem examinations in patients previously repeatedly examined while living is much greater than has been published.

DR. S. MACCUEN SMITH, Philadelphia: My feeling is that what might be termed classic otosclerosis is uncommon. I mean by this the type of case which is definitely hereditary, in contradistinction to those which are perhaps first hypertrophic and then, through certain histologic changes, become atrophic or sclerotic. On a former occasion I referred to a distant relative by marriage in whose family, for several generations, otosclerotic changes were definite in all those members who "took after" the maternal side. At about the age of 16 every child who resembled, spoke like or exhibited any of the other traits of his maternal ancestor began to lose hearing and later developed all the characteristics of otosclerosis. It fell to my lot to try to prevent these changes in several of the children before they arrived at this age. They were sent abroad, placed under the care of competent men and spent as long as three years in countries of more or less elevation, with the view that climatic therapy, together with other lines of treatment, might stay or even prevent the onset of the sclerotic changes. In each instance the result was just about the same, although perhaps slightly delayed, as it would have been had they remained in this country.

DR. JAMES G. DWYER: I agree with Dr. Duel as to the question of heredity in otosclerosis. I have seen three cases of true otosclerosis in six years, all hereditary, following the mendelian laws, and I think that the history is the most important point in these cases. I want to congratulate Dr. Emerson, who was the first to bring this point forward. I agree with Dr. Fowler that to make a diagnosis we must make a description that will not be misleading. I think that the textbooks are misleading and that most of the descriptions are wrong. It is only when the audiogram is combined with all the other facts that it is of aid in making a differential diagnosis. As to the last speaker, let me say that we should make comparative hearing tests, and base our ideas on them.

Thursday Afternoon Session, May 23, 1929

HEARING FROM THE PSYCHOLOGIST'S POINT OF VIEW. PROF. MADISON BENTLEY, Cornell University.

Helmholtz long ago supplied a convenient and satisfactory account of the ear and its auditory functions in his "resonance" theory, but in the last two or three decades the facts of audition have been greatly increased and the knowledge of the organic functions considerably enlarged. The facts are now so complex that the older and the simpler theories no longer suffice. In order to construct an adequate theory, therefore, it is necessary for anatomist, physiologist, embryologist, psychologist, neurologist, acoustical engineer and comparative morphologist to labor cooperatively and with an intelligent understanding of each other's problems and methods.

For his part, the psychologist, who is with all the others interested in the living organism, specifically directs his attention to the experience of hearing sounds. That experience he seeks to describe and to understand. To this end: (1) he describes the simpler auditory experiences, the tones, formants and noises, (2) he looks for the relation of these experiences to the forms of energy delivered at the cochlea and to the aural structures and mechanism and (3) he accounts for such complex auditory phenomena as combination tones, beats, fusions, binaural synergies and spatial localization of sounding objects. In all of these undertakings his problems and his methods supplement those of the other sciences and arts which conjointly attempt to understand hearing and to preserve the acoustical

functions of the organism. Although the facts, when they are gathered from all the sources, are extremely tangled and complicated, the present tendency to forsake traditional formulas and to work cooperatively in the laboratories and the clinic promises to provide us, before long, with an adequate and satisfactory doctrine of hearing and of its defects.

DISCUSSION

DR. ROBERT SONNENSCHEIN, Chicago: I am neither a psychologist nor a physicist but there is a question that I should like to ask Dr. Bentley regarding one of the points he covered. Is there any analogy from a physical standpoint between the loudness and brightness of the high tones as compared with the dulness and softness of the low tones? We know that by air conduction, we hear the sounds of the prongs of the tuning fork which have transverse vibrations of very little intensity but great amplitude. On the other hand, vibrations of the stem which we usually hear by bone conduction are longitudinal, of great intensity but very little amplitude. When the vibrations of the stem, however, are conducted through the lumen of a tube and ear piece by way of air conduction, as in the Sonnenschein-Minton modification of the Rinne test, these sounds are heard just as loudly as those of the prongs. Is it possible that while the same amount of energy is being used, it is different in quality, the low tones being dull and the high tones being bright and loud?

PROF. MADISON BENTLEY: It is through the magnanimity of the chairman that I gave the psychologist's point of view of the problem involved in hearing. In order not to propound anything too revolutionary I have kept the manuscript in a folder marked "antiques."

DR. STACY R. GUILD, Baltimore: I should like to congratulate Dr. Bentley on his presentation, and to add some qualifying phrases regarding our knowledge. Let me point out that structure comes before function. We are weak in our knowledge of fundamental things, and this fact has depressed me for a long time. We do not know yet how many nerve fibers of the cochlear division of the nerve have terminals on one hair cell. We do not know whether there are one, two or fifty having collaterals ending on that hair cell. We do not know how many hair cells receive collaterals from one and the same neuron; if fifty, that neuron can be stimulated by fifty impulses. In regard to transmission, when we start back from the organ of Corti to the cerebral cortex we have no idea how many cells in the cerebral cortex are stimulated by one cell in the cochlear spiral ganglion. We know that there is a dorsal and a ventral neuroganglioniculate body; but we do not know whether one spiral cell goes to one cortical cell, or whether one cortical cell receives impulses from one spiral cell only. It may be known, but I have not found any evidence worth printing that tells anything about this fundamental investigation.

DR. J. GORDON WILSON, Chicago: I think that the reason we do not get results is because our point of view is not the same. Dr. Bentley as a psychologist, coming to a medical meeting, has an entirely different point of view from ours, and it will do no harm for us to try to see things from his angle. The paper is scholarly, full of accumulated facts, but most of us will have to study it carefully to appreciate it.

PROF. MADISON BENTLEY: I cannot trespass on the field of anatomists and physicists. The incompleteness of the anatomic side of the paper is evident. It is extremely difficult to find out whether one receptor is connected with several fibers or whether one fiber may not be associated with several nerve cells. Until we can get a simple case we shall not be able to make out this complicated mechanism in the study of the ear.

LIMITED LESIONS OF THE BASILAR MEMBRANE. DR. EDMUND PRINCE FOWLER, New York.

This article appears in full in this issue, p. 624.

DISCUSSION

DR. STACY R. GUILD, Baltimore: I should like to ask Dr. Fowler one question, What is his evidence that the 7 mm. point corresponds to the 4,000 pitch in the human being? I am sure that high pitches are heard at the lower basal end, and low tones at the apical end where the membrane is broader. I am trying to arrive at a conclusion as to how sound waves become converted into energy, but I do not know yet.

DR. EDMUND P. FOWLER: This is a theoretical arrangement as to the division of the basilar membrane into frequency spaces, and it is about at that distance that theoretically the 4,000 vibration would occur.

DR. STACY R. GUILD: There is no observed evidence that the theoretical point is correct.

DR. EDMUND P. FOWLER: We have endeavored to produce this in animals, but so many unexpected things can happen to laboratory animals. We often have to begin all over again. We have a new set of ears from animals going through the decalcifying solutions now.

DR. STACY R. GUILD: In regard to the distribution of the gun shot injury in the guinea-pig, it does not fall at that point. In the guinea-pig the membrane is 18 mm. long and the area of deafness does not occur in the same place. I used sixty animals in the tests. One ear was exposed and one covered. When plotting out the injured area it was found occasionally as far down as the vestibule.

DR. EDMUND P. FOWLER: This 7 mm. point is suggested from theoretical reasoning, but any other point would be equally satisfactory. In the human subject we have to wait a long time for suitable cases for autopsy, and when we have obtained enough cases with these gaps and loops, and then have had them examined post mortem, we shall be able to speak with more certainty on this question of the 4,000 vibration point.

DR. STACY R. GUILD: With the audiometer one man heard 2,048 but did not get 4,096. He was not checked for the intermediate pitches but he fell off somewhere between the two points. In this ear, degeneration was distinct. There was a total atrophy of the organ of Corti and of nerve fibers in the spiral laminae at the 9 to 10 mm. point. Because the lower half of the basal turn is much longer than the rest we also did monochord tests. The man heard up to 13,000 on the audiometer. When we examined the section there was atrophy, yet he was hearing 13,000. He had normal nerves at the organ of Corti, but he had a patch of degeneration.

DR. A. G. POHLMAN, St. Louis: The first work done on quantitative bone transmission was that by Dr. Kranz and myself. We reported certain lowered acuity around 2,600 double vibrations. I had a case of lowered acuity which corresponded to Dr. Fowler's tone gaps. The next person tested was normal on one side, and had a total tone gap for bone transmitted sound on the other. The question was why he did not hear this with the better ear. Air transmission was normal, but there was a hole in bone transmission. I have the record of a case that was seen by us in which the bone acuity was taken before and after treatment, and showed a distinct area of lowered acuity for bone transmitted sound. Later the bone transmitted sound gap disappeared. We thought that it was a localized lesion of the basilar membrane. It is perhaps somewhat beside the question, but it seems that some of these lowered areas shift and sometimes they disappear, which shows that they are not a permanent lesion.

DR. D. W. DRURY, Boston: I wish that I had some power with the medical examiner to insist on autopsy of cases that show these types. It is a most interesting problem. I have seen them develop by audiometer records. I think that if these cases could be checked up by autopsy it would make a great advance in our knowledge.

DR. ARTHUR DUEL, New York: I should like to recount a phenomenon which recently came to my attention. It was the case of a well known pianist who came for treatment for furunculosis of the ear. He became interested in the tuning fork tests. He told me that he had perfect pitch perception by which he could always tell when the violin or piano was in perfect tune. He said that he was able to determine this because he knew the pitch was actually always exactly one-third lower than it seemed. In his mind he always transposed this third and so arrived at the correct pitch. I verified this with the Bezold-Edelmann series of tuning forks.

DR. C. C. BUNCH, Baltimore: The audiogram I showed was made from tests of a young lady in whom the right ear was practically normal. Low tones up to 256 double vibrations were normal in the left ear but at 512 double vibrations there was considerable loss and this increased for tones of higher pitch, the greatest loss being at 1,448 double vibrations. She heard 4,096 but did not hear 6,793 double vibrations at their maximum intensities. Double vibrations of 10,321 were heard but 13,004 and 16,384 were inaudible. With the monochord by bone conduction, she could hear 10,000 but could not hear 9,000 or 11,000 double vibrations. A case like this brings up several interesting problems concerned with theories of hearing. Do these people actually hear sounds at their proper pitch or is there false perception? In this case, low tones were heard at their true pitch when the two ears were compared, but the tone of 2,896 double vibrations seemed in the left ear to be lower than 1,024 but higher than 724. It seems difficult to account for this false hearing on the basis of any theory of hearing.

PROF. MADISON BENTLEY, Cornell University: The reference to tinnitus reminds me that Stumpf, the psychologist, had a method for determination of the pitch of the tinnitus. Dr. Fowler's reference to the anatomic conditions in his test interested me and reminded me of research done on guinea-pigs with long-continued exposure to carefully controlled rates of stimulation. The result of stimulation seemed to be dependent on the rate of the continued stimulus, as if the rate were the determining factor. If we could go back and determine the character of the sounds, and if this were known, we should know the reason for sudden and continued tinnitus, whether or not this was the proper vibration rate for the tympanum. We might discover whether or not there is a certain region of the scale that has a certain sensitivity to shock because of the relation of the tympanic membrane to this frequency.

DR. M. A. GOLDSTEIN, St. Louis: Another phase of this problem is the relation of the peripheral hearing mechanism to the central hearing mechanism. We ascribe too much importance to the analytic ability of the cochlea or the organ of Corti. For some years I have insisted on tone islands and their recognition through studies by the audiometer. It took me some years to convince physicists that there were tone gaps. I said that there was a limitation of the audiogram. I established an arbitrary line, that is, the line of serviceable hearing, and applied that in testing the availability of the audiometer. My claim is that in certain types of serious deafness one may find a hearing capacity below the ability to which the audiometer has been constructed. When one gets a tone perception below this limitation or arbitrary curve of serviceable hearing, one cannot tell whether the patient hears that sound or feels it. It is difficult to determine where hearing ends and where touch begins. In the work on reeducation, when there are tone gaps in the scale, gaps of hearing obtain and these gaps can be reconstructed. We know that nerve tissue is the last tissue of the body to regenerate. We do not get frequencies in the cochlea that are lost, but we can get moderate reeducation of sounds that are lost. This answers the question that Dr. Duel raised in regard to this mental sound that this professional musician carried in his brain. There are a mechanically perfect machine and an educated sense in the auditory field and these cooperate in the whole problem of hearing. We should not confine ourselves entirely to the consideration of the peripheral mechanism.

DR. GEORGE SHAMBAUGH, Chicago: This phenomenon is one of the most interesting sidelights on the physiology of hearing and sound perception. The audiometer, as I use it, has its greatest value in throwing light on physiology, rather than in making diagnoses. Recently, in Chicago, I saw a patient with this phenomenon—three weeks before, quite suddenly in the middle of the day she had an attack of tinnitus aurium; the ear felt full and there was a defect in hearing. One ear was normal; the other ear had a tone range from 512 to 1,024 double vibrations, and then dropped off immediately. My interpretation was that she heard nothing above that point. What had happened to that woman? She had not heard a gun shot; she did not use drugs; she had no toxemia. I think that it was a circulatory accident, and that an embolus lodged in the labyrinthine artery. That sort of thing might give one some clue to where the point is that the artery strikes the basal coil. Dr. Fowler spoke of those gaps not being found in otosclerosis, but I think that they do occur often in that condition.

DR. J. HOLINGER, Chicago: This discussion brings to my mind a patient whose observations I presented at the International Congress in Boston. It was a case of diplocusis. The patient was a musician, who heard a different pitch in each ear. I examined him by means of two forks, one held before each ear the pitch of one being changed until the patient heard consonance. The deviation of both ears changed with the pitch, being greatest in the middle octaves. Of course it is a disease of the nerve, but whether labyrinthine or central is questionable. Diplocusis disables a musician entirely.

DR. J. GORDON WILSON, Chicago: I should like to ask a question. Ten years ago I was interested in that drop. I found that it had a relation to the tinnitus. I thought that the position of the tinnitus was higher. I found it nearer the top than the bottom.

DR. J. A. BABBITT, Philadelphia: I feel some hesitation in presenting a practical phase on the clinical side of Dr. Fowler's excellent paper, as the previous discussion has been entirely technical. I should like, however, to report a case corroborating his emphasis on the value of routine tests.

After a long period of treatment by various specialists this patient came to me with the single pronounced ailment of tinnitus. He was studied by various departments and was seen by one of our associates, Dr. Coates. He was investigated by internists, neurologists, the Bárány people and had had many laboratory tests. An extensive intranasal pathologic process was corrected, all to no avail. An outstanding feature in his case, however, was a curious rise of 20 and drop of 40 sensation units in the audiogram at 2,048 and 4,096 double vibrations, respectively, and this was sustained through all subsequent studies. It was limited to the right side, the side of the tinnitus, and corresponded in general to our analysis of the pitch of his tinnitus though his tuning fork reaction was untrustworthy. It seemed worth while to record this in the discussion as all the evidence seemed to point toward a limited basilar membrane lesion.

DR E. P. FOWLER, New York: Dr. Guild's question is interesting. The 4,000 vibration point is a rough approximation. I also suggested above 1,000 to make it as broad as possible. To carry out the hypothesis of hearing, my understanding is that it is not necessary to have the proximal part of the cochlea intact in order to hear the upper notes. There are two ways in which these notes can be supplied in event of anatomic and pathologic failure of the proximal part of the basilar membrane. First, if you take a note and cut off its fundamental, the brain will still give the fundamental, or the mechanism of the ear reproduces it. Second, if the upper note areas are missing, there may be a redistribution of the frequencies along the cochlear membrane. If the upper note areas are absent the next lower areas may be able to function to supply partially the deficiency. The basilar membrane is sometimes bony, not membranous, near the vestibule, and yet the tones of the short wave lengths below may be heard well. Other tones may be heard because of the overtones. In the case Dr. Bunch cites, the area tones were not heard because the tones just below them were not

heard loudly enough. Dr. Pohlman used the continuous frequency apparatus. I understand that this is not as reliable as the audiometer (I may be wrong), but this would explain the variability in results. I tried for a long time to get a continuous tone audiometer, but the Western Electric people were unable to make it at that time.

In regard to the change of tone sensation, with the limited work I have done, I find it is difficult to get untrained observers to distinguish between small changes of intensity and changes of frequency. They mistake one for the other and this easily leads one astray. In regard to Dr. Babbitt's patient, in whom there was a good deal of nasal pathologic change, let me say that many patients do have sinus trouble, and tinnitus may sometimes arise from nasal pathologic change. Often bad teeth cause tinnitus. One may get embolism with acoustic shocks, but I believe that a simpler explanation would be embolus of the proximal branch of the artery. That would cut off the upper frequencies entirely. Some one mentioned the keynote of the basilar membrane. It is a very high note much above the frequency point under discussion. Dr. Goldstein mentioned limits of hearing—we had to stop our tests of the upper limits or get into legal troubles because of possible damage to hearing. Whether or not one can make use of the audibility below the point of serviceable hearing is a question of degree. Above a certain intensity although there is much sound, there is little definition. Several cases were sent down to us by the Wright Aural School. These children had residual hearing and the question was whether we could teach them to speak. If a child has even slight residual hearing it should be utilized to the utmost. This is an important point in the teaching of the very deaf.

The first case I showed had diplocusis in one ear, and it was lost about half way up the slope. I did not examine all of the cases for the exact location of the tinnitus. It takes a long time to obtain this information. I examined twelve patients, and in some it was on the lower slope, in some on the upper slope. Often the patients confuse frequency and intensity. If we could get beats, we could get better information as to what is going on in the organ of hearing. In but one or two instances have we been convinced that the phenomenon of beats was obtained.

PRELIMINARY NOTE ON THE USE OF NEGATIVE PRESSURE IN THE DIAGNOSIS AND TREATMENT OF DEAFNESS. DR. J. F. FAIRBAIRN and DR. A. G. POHLMAN, Buffalo.

DR. POHLMAN said that it is well known that prolonged occlusion of the auditory tube results in a progressive loss of acuity of hearing accompanied by retraction of the drum membrane. Some years ago Pohlman pointed out that the degree of hearing loss and the amount of drum membrane retraction were entirely out of proportion to the negative pressure developed in the closed middle ear. He suggested that the tensor tympani muscle gradually adjusted itself to the slight displacement of the drum membrane (took in the slack as it were) and that this tonus contracture of the muscle with or without further adhesion damped out the vibrations of the drum membrane. He also mentioned the operative possibility of lengthening the contracted muscle by clipping off the processus cochleariformis. The development of this operative technic led to the idea of creating a negative pressure in the external canal to pull the drum membrane lateralward and thereby put the muscle on the stretch and loosen the parts through fatigue of the muscle. It must be remembered in dealing with sound that the usual amplitudes heard are ultra-microscopic displacements and that a loosening effect equivalent to one hundred-thousandth of an inch should be sufficient to make a difference in the intensity required to reach the threshold of hearing.

The patient is first carefully tested for air acuity. The authors employed a beat frequency oscillator with a standard audibility meter shunted across the receiver. The oscillator pitch is plotted at every fifth mark on the dial which

gives sixteen intervals in the range from 120 to 8,300 Hertz. The audibility motor gives thirty-three graded steps of intensity. Next a glass tube is sealed in each external auditory canal with dental impression compound and the entire auricle filled with plaster-of-paris. The patient is then subjected to a negative pressure of from 30 to 40 cm. of water controlled by a water manometer for ten minutes with occasional oscillation in the pressure of from 10 to 20 cm. The seals are then removed and the acuity of hearing checked. The seals may be used again for a second and all subsequent treatments by applying a liberal coat of petrolatum and reinserting. The second treatment is made with a slightly higher pressure of from 60 to 80 cm. of water, and it has been the essayist's experience that a greater negative pressure is not required.

Seventy-nine patients with deafness have been treated in our office in Buffalo with improvement in thirty and subjectively obvious improvement in twenty-one persons.

"It is impossible from the limited number of cases studied thus far to determine in advance which type of case will improve and which type will not improve. Neither is the procedure absolutely fool proof."

In a general way patients who report a paracusis Willisii and who respond positively to the acoustic fan test and those with functionally patent tubes seem to react the best. It is also interesting to note that in several cases the low pitch head noises have been completely eliminated although so far we have found no improvement in alleviating the high pitch sounds.

It is not unlikely that the patients who show every evidence of excellent end-organs by reason of the range and acuity of the bone transmitted sound determined with a bone activating receiver are suffering from some sort of mechanical disturbance in the sound transmission system in which the delicate negative pressure is not effective.

DISCUSSION

DR. H. H. VAIL, Cincinnati: I have attacked the same problem from a different point of view, but my work is still in the early experimental stages. We have blocked up the external auditory canal with modeling wax and have subjected the animal to compressed air in a compression tank to get positive pressure in the middle ear rather than negative pressure which has been described by Dr. Pohlman. Before trying it on my patients I want to see what happens to the middle ear. One rabbit showed small hemorrhages in the mucosa of the middle ear. The treatment that has been outlined by Dr. Pohlman has opened up a new field which should be investigated. I know that some cases must have adhesions which we can break up by pressure better than by massage.

DR. GEORGE SHAMBAUGH, Chicago: Were these all cases of retracted drum membrane?

DR. A. G. POHLMAN: One cannot tell which patients will be improved. I have had good response in people in whom I did not expect to get any result and vice versa. In most cases there was a retracted and atrophic membrane. In many cases there was paracusis.

DR. D. HAROLD WALKER, Boston: In these cases the tensor tympani muscle is an important factor. Occlusion of the eustachian tube is not sufficient to account for the retraction of the drum membrane alone. The narrowing of the middle meatus is important also. When the middle turbinate is squeezed between the nasal wall and septum, this condition may cause a spasm of the tensor tympani muscle.

DR. J. F. FAIRBAIRN, Buffalo: These cases have been investigated thoroughly. In seventy-six there was positive infection in the right antrum and considerable treatment had to be carried on. In cases in which the acoustic insult had been caused by a pneumatic drill we found nothing particular in the nose or throat.

SOME OBSERVATIONS ON ANATOMIC INSTRUCTION FOR GRADUATE STUDENTS WHO ARE PREPARING FOR PRACTICE IN THE FIELD OF OTOLARYNGOLOGY. DR. GEORGE E. SHAMBAUGH, Chicago.

Dr. Shambaugh believes that there is a rapidly increasing tendency toward specialization, so that the problem of providing facilities for men who want to enter our particular specialty stares us in the face. Undergraduate medical instruction is stabilized although it could be improved, but the preparation for actual practice of the specialty presents more difficulties. It is not taken up by the university as it should be, with the result that most men are not adequately prepared. The fact of doing a few operations on the cadaver is no preparation at all. Since the World War Dr. Shambaugh has undertaken to see what we can do during the first year in the day of fundamentals. There are two problems to be met: (1) to train men to make proper examination so as to build up sufficient background of clinical experience in order that they may know how to recognize indications for treatment, especially indications for operative intervention. (2) The second is the more difficult problem, that is, trying to build up for these men an adequate knowledge of the fundamentals in anatomy. This is essential if the man is to be a successful, safe practitioner in otolaryngology. Giving him all sorts of bone to operate on is not enough, because the surgical points of interest cannot be picked up.

Dr. Shambaugh's plan of instruction is to take twelve men, three each quarter, and have them pay a fee for the supervision. They are employed as clinical assistants. If they pay in advance we are sure that they will not look for other positions in the middle of the course. Half the day is spent in supervised clinical study of patients. The anatomic training consists in the first quarter year in doing a careful dissection of the head and neck; in the second they make preparations of sections; in the third and fourth quarters they proceed with embryologic and microscopic preparations. The work is different from such work among undergraduates, with whom it is more or less perfunctory. These men grasp every opportunity to work with anatomic preparations. Dr. Shambaugh has found that the best way to impress ideas on a student's mind is to let him make a drawing, for in this way the idea becomes graven on his memory.

DISCUSSION

DR. EDWARD B. DENCH, New York: Anything that will fix the anatomy of the ear in the minds of medical students is a distinct advance in medical education. I know that I had considerable difficulty in fixing the gross anatomy of the ear in the minds of my own students. After carefully explaining to them the importance of understanding the normal anatomy of the parts, I give them some operative work on the cadaver, having them demonstrate to me the mastoid operation. In almost every case the students would disregard what had been said about the anatomic landmark for the mastoid antrum and would expose the lateral sinus, or enter the middle cranial fossa. The method which Dr. Shambaugh has suggested of having the men make tracings in the study of the temporal bone in excellent. It is difficult to copy a microscopic specimen, but it is easier to do than the gross specimen. I should like to bring out here a point that I emphasized in discussing this paper in New York. Sir William MacEwen, in his book "Diseases of the Brain and Spinal Cord," mentions a space known as MacEwen's triangle. A much simpler method of locating the mastoid antrum, it seems to me, is by a triangle known as the Dench triangle. If a horizontal line is drawn tangent to the superior border of the bony external auditory meatus, the aditus ad antrum lies within the triangle the two sides of which are formed by the superior and posterior tangent lines and the base of which is formed by the curvilinear line of the bony external auditory meatus, included between these tangents. This will locate the mastoid antrum in every instance. The more the operator invades the posterior line, the more likely he is to enter the lateral sinus. The more he invades the superior line the more he is endangering the middle cranial fossa.

DR. D. HAROLD WALKER, Boston: In Boston, the Harvard University does not control the Eye and Ear Infirmary. We no longer give a detailed clinical course, but do give a thorough anatomic and pathologic one. Dr. Mosher has limited his work to anatomic studies. He is trying to do as Dr. Shambaugh does, to teach by models. He allows the students to make plaster casts in order to fix the anatomic details in their minds. Dr. Dench's remarks on the antrum are interesting and embody good suggestions. There is a great danger to the facial nerve by going too near the fallopian canal, along the posterior canal wall.

DR. S. MACCUEN SMITH, Philadelphia: I know of nothing more important than a proper anatomic foundation for those who are preparing for practice in the field of otolaryngology. Dr. Shambaugh's paper is, therefore, especially interesting and timely. We must all agree with him that it is impossible to learn the surgical anatomy of the ear simply by the study of dry bones, but that this must be followed by work on the cadaver and this in turn by work under careful observation and instruction on the living subject. I think that all of us who are conducting large clinics should make it a rule to require our assistants to pass the Board of Otolaryngology, and I further believe that we should not take on new men until this requirement has been met.

DR. L. W. DEAN, St. Louis: I think that we can all agree that one of the greatest things that Dr. Shambaugh has done for otology is in teaching the student that fundamentals should be thoroughly known before men can begin clinical work. In our work we have the students make drawings of the preparations and if there is a case of peculiar interest we let them make plaster casts also. I think that this is an important phase of the work and should have some supervision.

DR. GEORGE E. SHAMBAUGH: I spent some time in the infirmary in Boston and saw the models that Dr. Mosher is using in teaching. They were excellent. We do endeavor to give instruction as well. Dr. Sonnenschein teaches the students the tuning fork and other functional hearing tests. An operative course on the cadaver follows the anatomic course. We do not allow men in the operating room until the third quarter. They want to watch operations, and would like to operate the first day they get there. We tell them that we are not interested in allowing men to operate who do not appreciate the indications.

(To be concluded)

Book Reviews

MASTOIDS. By FREDERICK M. LAW, M.D. Volume I of Annals of Roentgenology. Second edition. Price, $12. New York: Paul B. Hoeber, Inc., 1929.

This book is an atlas of thirty-three full page plates made up of halftone reproductions of the mastoid, taken in health and in disease. The author brings to this work a knowledge based on extensive experience as a roentgenologist, and as a practicing otologist. In his long experience in the Manhattan Hospital, he has had an exceptional opportunity for following cases to the operating room, and in this way accumulating an extensive background in experience for the intelligent interpretation of roentgen observations.

The volume will serve as a valuable guide to the practicing otologist in undertaking to evaluate x-ray pictures of the mastoid.

The author emphasizes that final judgment of a case can usually be reached only by cooperation between the otologist and the roentgenologist. He also brings out that, for practical purposes, a single film will furnish the needed data, thus rendering unnecessary stereoptican films.

As a roentgenologist he does not advocate advising for or against operation, leaving this decision rightly for consultation with the otologist.

In separate chapters, he discusses the observations on: mild acute mastoiditis; acute purulent mastoiditis; severe acute purulent mastoiditis; acute purulent mastoiditis with abscess formation; cholesteatoma; mastoids with large emissary vein.

The technic of making mastoid plates is carefully described, including the conditions necessary for the proper exposure, as well as the development of the film.

Directory of Otolaryngologic and Ophthalmologic Societies *

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Robert C. Lynch, Maison Blanche Bldg., New Orleans, La.
Secretary: Dr. Gabriel Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Place: Detroit. Time: June 23-27, 1930.

American Bronchoscopic Society

President: Dr. Thomas E. Carmody, Metropolitan Bldg., Denver.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

American Laryngological Association

President: Dr. Lewis A. Coffin, New York.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

American Laryngological, Rhinological and Otological Society

President: Dr. Ross Hall Skillern, 1811 Spruce St., Philadelphia.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.
Place: Atlantic City, N. J. Time: May, 1930.

American Otological Society

President: S. MacCuen Smith, Philadelphia.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Chicago. Time: October, 1930.

LOCAL

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. Harold S. Muckleston, 6422 Hollywood Blvd., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of each month.

The Colorado Oto-Laryngological Society

President: Dr. Frank L. Dennis, Ferguson Bldg., Colorado Springs.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg., Time: First Saturday of each month from October to May.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Louis S. Greene, 1710 Rhode Island Ave. N. W., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

Atlanta Academy of Ophthalmology and Otolaryngology

President: Dr. H. F. McDuffie, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. Leon E. Brawner, 157 Forrest Ave. N. E., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. George B. Smith, Rome.
Secretary-Treasurer: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.

Chicago Laryngological and Otological Society

President: Dr. Samuel Salinger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Albert E. Bulson, 406 W. Berry St., Fort Wayne.
Secretary: Dr. Robert J. Masters, 601 Hume-Mansur Bldg., Indianapolis.
Place: French Lick Springs Hotel. Time: Dec. 12-13, 1929.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. Robert J. Masters, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

New Orleans Ophthalmological and Otological Society

President: Dr. M. Earle Brown, Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., third Thursday of each month from September to June.

Baltimore City Medical Society, Section on Otology and Laryngology

Chairman: Dr. Waitman F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Moses L. Breitstein, 1212 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

New England Otological and Laryngological Society

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary-Treasurer: Dr. Charles T. Porter, 520 Commonwealth Ave., Boston.

Detroit Oto-Laryngological Society

President: Dr. C. C. McClelland, David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. Don A. Cohoe, 13535 Woodward Ave., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

The Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. N. Spratt, 900 Nicollet Ave., Minneapolis.
Secretary-Treasurer: Dr. J. H. Morse, 80 S. 7th St., Minneapolis.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. L. R. Forgrave, Logan Bldg., St. Joseph, Mo.
Secretary: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. F. C. Simon, Arcade Bldg., St. Louis, Mo.
Secretary: Dr. M. L. Hanson, Murphy Bldg., East St. Louis, Ill.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7 p. m., third Wednesday of each month from October to May.

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frederick J. Wort, 1080 Broad St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

New York Academy of Medicine, Section of Laryngology and Rhinology

Chairman: Dr. Mervin C. Myerson, 12 E. 86th St., New York.
Secretary: Dr. John M. Loré, 240 West End Ave., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

New York Academy of Medicine, Section of Otology

Chairman: Dr. Clarence H. Smith, 2 E. 54th St., New York.
Secretary: Dr. James Garfield Dwyer, 57 W. 57th St., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. John P. O'Keefe, 251 State St., Albany.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. A. Vincent Walker, 833 South Ave., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George M. Constans, Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Harris H. Vail, 24 E. 8th St., Cincinnati.
President-Elect: Dr. Edward King, Pearl Market Bank Bldg., Cincinnati.
Secretary-Treasurer: Dr. Donald Lyle, 19 W. 7th St., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Myron Metzenbaum, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Arthur C. Jones, 105 N. 8th St., Boise, Idaho.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

PHILADELPHIA LARYNGOLOGICAL SOCIETY

President: Dr. Henry A. Laessle, 13th and Spruce Sts., Philadelphia.
Secretary: Dr. Herman B. Cohen, 13th and Spruce Sts., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLOGY AND LARYNGOLOGY

Chairman: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Clerk: Dr. Frederick M. Strouse, 13th and Spruce Sts., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

PITTSBURGH OTOLOGICAL SOCIETY

Chairman: Dr. John R. Simpson, 115 South Highland Ave., Pittsburgh.
Secretary-Treasurer: Dr. Watson Marshall, 121 University Place, Pittsburgh.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. J. E. Raia, 272 Broadway, Providence.
Secretary: Dr. F. W. Dimmitt, Jr., 195 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William Thornwall Davis, 927 Farragut Sq., Washington, D. C.
Secretary: Dr. Fletcher D. Woodward, Box 162, University, Va.
Place: Miami, Fla. Time: Nov. 20-22, 1929.

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. R. J. Warner, 706 Church St., Nashville.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. D. L. Bettison, Medical Arts Bldg., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, Medical Arts Bldg., Dallas, Texas.
Recording Secretary: Dr. L. A. Nelson, 4105 Live Oak, Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. C. P. Schenck, Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. John H. Foster, 1300 Walker Ave., Houston, Texas.
Secretary: Dr. Edward W. Griffey, Houston Clinic, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. F. W. Sorrell, Gibbs Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. A. F. Clark, 728 Main Street, San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

Utah Ophthalmological Society

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Ezra Thompson Bldg., Salt Lake City.

Virginia Society of Otolaryngology and Ophthalmology

President: Dr. H. B. Stone, 303 First St., Roanoke.
Secretary-Treasurer: Dr. Fletcher D. Woodward, Box 162, University.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Will Otto Bell, Medical-Dental Bldg., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. O. M. Rott, Paulsen Bldg., Spokane, Wash.
Secretary: Dr. C. A. Veasey, Jr., 421 Riverside Ave., Spokane, Wash.

The Milwaukee Oto-Ophthalmic Society

President: Dr. L. P. Allen, 145½ Main St., Oshkosh, Wis.
Secretary-Treasurer: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

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